A Dissertation on

# "A STUDY OF ETIOLOGICAL ANALYSIS OF ACUTE PANCREATITIS AND THEIR OUTCOME IN PATIENTS ADMITTED AT RGGGH"

Dissertation submitted to

**THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERISTY**

**CHENNAI**

with partial fulfilment of the regulations

for the Award of the degree

**M.S. (General Surgery)**

Branch – I

**MADRAS MEDICAL COLLEGE ,**

**CHENNAI.**

**APRIL-2015**

# BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of Dr.K.J.VIGNESVARAN on "A STUDY OF ETIOLOGICAL ANALYSIS OF ACUTE PANCREATITIS AND THEIR OUTCOME IN PATIENTS ADMITTED AT RGGGH" during his M.S. (General Surgery) course from March 2014 to August 2014 at the Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai – 600003.

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## ACKNOWLEDGEMENT

I would like to express my deep sense of gratitude to the Dean, Madras Medical College and Prof.Dr.P.RAGUMANI M.S, Director, Institute of General Surgery , MMC & RGGGH, for allowing me to undertake this study on “A STUDY OF ETIOLOGICAL ANALYSIS OF ACUTE PANCREATITIS AND THEIR OUTCOME IN PATIENTS ADMITTED AT RGGGH”

I was able to carry out my study to my fullest satisfaction, thanks to guidance, encouragement, motivation and constant supervision extended to me, by my beloved Unit Chief Prof. Dr. A.RAJENDRAN, M.S. Hence my profuse thanks are due for him.

I am bound by ties of gratitude to my respected Assistant Professors, Dr.A.Sagaya inba sekar, Dr.P.Ramadoss and Dr.Gajendra Raj,Dr.Selvakumar in general, for placing and guiding me on the right track from the very beginning of my career in Surgery till this day. I would be failing in my duty if I don’t place on record my sincere thanks to those patients who inspite of their sufferings extended their fullest co-operation.

I am fortunate to have my postgraduate colleagues Dr.PadamKumar, Dr.Kondba Magade, Dr.Arunkumar, Dr.Sethu Kannan ,Dr.Selvaraj for their invaluable suggestions, relentless help for shouldering my responsibilities. Simply words cannot express its depth for their unseen contributions. Lastly, my lovable thanks to my parents for their moral support.

# DECLARATION

I , certainly declare that this dissertation titled, "A STUDY OF ETIOLOGICAL ANALYSIS OF ACUTE PANCREATITIS AND THEIR OUTCOME IN PATIENTS ADMITTED AT RGGGH", represent a genuine work of mine . The contribution of any supervisors to the research are consistant with normal supervisory practice, and are acknowledged.

I , also affirm that this bonafide work or part of this work was not submitted by me or any others for any award , degree or diploma to any other university board , neither in India or abroad . This is submitted to The Tamil Nadu Dr.MGR Medical University, Chennai in partial fulfilment of the rules and regulation for the award of Master of Surgery Degree Branch 1 (General Surgery).

Date :

Place:

Dr.K.J.VIGNESVARAN

# INSTITUTIONAL ETHICS COMMITTEE
## MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
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## CERTIFICATE OF APPROVAL

To

**Dr.K.J.VIGNESVARAN,**
Post Graduate,
Institute of General Surgery,
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Chennai - 600 003.

Dear **Dr.K.J.VIGNESVARAN,**

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled **"A Study of Etiological analysis of acute pancreatitis and their outcome in patients admitted at RGGGH"** No.490614.

The following members of Ethics Committee were present in the meeting held on 03.06.2014 conducted at Madras Medical College, Chennai-3.

| | | |
|---|---|---|
| 1. | Dr. C.Rajendran, M.D, | -- Chairperson |
| 2. | Prof. Kalaiselvi, M.D,<br>Vice Principal, MMC, Ch-3 | -- Member Secretary |
| 3. | Prof. Nandhini, M.D,<br>Inst. of Pharmacology, MMC, Ch-3 | -- Member |
| 4. | Prof.G.Muralidharan, M.S,<br>Prof & HOD General Surgery, MMC, Ch-3 | -- Member |
| 5. | Prof.V.Padmavathi, M.D,<br>I/c. Director of Pathology, MMC, Ch-3 | -- Member |
| 6. | Thiru. S. Govindasamy, BA., BL | -- Lawyer |
| 7. | Tmt.Arnold Saulina, MA MSW | -- Social Scientist |
| 8. | Thiru.S.Ramesh Kumar,<br>Administrative Officer, MMC, Ch-3. | -- Lay Person |

We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLE

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# LIST OF ABBREVATIONS USED

| | | |
|---|---|---|
| ICU | → | Intensive care unit |
| CBD | → | Common Bile Duct |
| APACHE | → | Acute Physiological And Chronic Health Evaluation |
| CT | → | Computer Tomography |
| ARDS | → | Acute Respiratory Distress Syndrome |
| DIC | → | Disseminated Intravascular Coagulation |
| SAPS | → | Simplified Acute Physiological Score |
| MRCS | → | Medical research council sepsis score |
| CVP | → | Central Venous Vressure |
| MAP | → | Mean Arterial Pressure |
| NPO | → | Nil Per Oral |
| MRI | → | Magnetic Resonance Imaging |
| PCD | → | Per Cutaneous Drainage |
| FNA | → | Fine Needle Aspiration |
| TPN | → | Total Parenteral Nutrition |
| USG | → | Ultrasonography |

# ABSTRACT

## BACKGROUND AND OBJECTIVES

Acute pancreatitis accounts for 3% of all cases of acute abdomen it occurs in any age group but peak incidence is in young men and older women.

There are two major causes of acute pancreatitis-alcohol and biliary disease which accounts 50-70% of total cases. The pathological spectrum varies from edematous pancreatitis which is uncomplicated and self limiting to necrotizing pancreatitis in which degree of pancreatic necrosis correlate the severity of attack and systemic complication which involve renal, lung, git, brain and may lead to multi system organ failure.

## METHODS

It is hospital based prospective study. The study was conducted in hospitals attached Madras medical college during the study period from March 2014 to August 2014. 100 consecutive complicated cases were analyzed.

## RESULTS

Out of 100 patients 93 were male and 7 female. The peak incidence in male is $5^{th}$ decade and in female $2^{rd}$ decade in life. Alcohol accounts72% of total cases and 16% biliary disease.26 patients

found to have systemic complication and 58 local complications, most systemic complications were managed in general ward except few in ICU those associated with ARDS and septicemia. 34% of patient had recurrent attack supportive care in ICU for the period of 3 weeks, 80% resolved, infected necrosis were managed by necrosectomy, mortality 33%. Pancreatic abscess were managed by USG/CT guided aspiration.

## CONCLUSIONS

Acute pancreatitis is one of the common differential diagnosis of acute abdomen specially alcoholics and biliary disease. Patients with acute pancreatitis should be evaluated clinically, biochemically and radiologically as this condition associated with severe systemic and local complications


## KEY WORDS

Acute pancreatitis, pseudocyst, necrosis, abscess.

# TABLE OF CONTENTS

04

# INTRODUCTION

Acute pancreatitis has been recognized since antiquity[1,2] but the importance of pancreas and the severity of its inflammatory disorders were realized only in middle of 19th century[3].

The nature of disease was recognized way back in 1925 when Moynihan described acute pancreatitis as " The most terrible of all the calamities that occur in connection with abdominal viscera[4] " but even today with technical advantage in medical and surgical field acute pancreatitis remains a major cause of morbidity and mortality[5,6].

Acute pancreatitis is a common disorder. The data available are mainly from united states and united kingdom. It has been noticed in most of the studies that there is in an increase in the incidence of disease by a factor of 10 in the past 3 decades. The reason for the increase is speculated to be due to increase in alcohol abuse and an improved ability to diagnose the disease. But the disease has been a cause of significant morbidity and mortality. Both sexes are equally affected.

Acute pancreatitis is related to alcohol or biliary tract stone disease in 80% of Cases. The remaining 10% is related to metabolic

factor, drugs and other condition and 10% are idiopathic[3,6]. However the variation in the frequency of different forms of pancreatitis from source to source is quite varied and depends on country of origin and the population studied.

Acute pancreatitis has been classified way back in 1882 in to apoplectic and sub acute forms. Later in 1963 in a symposium at Marseilles a classifications based on the morphology was accepted until 1993 when it was realized that the earlier classification was possible only after surgery or during autopsy, so a classification system was very much needed at the outset of the disease for a working diagnosis. Finally a classification system, which is clinically based, was established in the Atlanta symposium 1n 1993[7].

Acute pancreatits is a pathological broad spectrum of disease ranging from parenchymal edema to severe necrotizing pancreatitis. Clinical presentations varies from mild abdominal discomfort to hypotension, metabolic derangement, sepsis, fluid sequestration, multiple organ failure and death. 9 of 10 experience mild to moderate course and self-limited, and 1 of 10 experience a severe life threatening form of acute pancreatitis. Based on the above it is presently classified into mild acute pancreatitis associated with minimal organ dysfunctions and uneventful recovery, and severe acute pancreatitis associated with

organ failure and \or local complications such as necrosis, abscess or pseudo cyst[7].

Diagnosis remain clinical and can be supported by 1.5 – 2 fold increase above the upper limit of normal of serum amylase[8]. But an estimation of serum lipase, trysinogen or isoamylase assay are confirmatory[8] and will increase the diagnostic yield. Supportive radiological procedure are sonography , computed tomography and MRI . Currently CECT is the imaging modality of choice where areas of hypo perfusion correlate with necrosis[9].

The treatment of acute pancreatitis is largely supportive. Patient with mild disease are treated by eliminating oral intakes, instituting intravenous hydration and providing frequent parenteral analgesia. Use of antibiotics and drugs, which reduce the pancreatic secretion, have been studied extensively[6]. Except for imipenem and somatostanin, which has shown benefit, no other drug have shown any promising result. For practical purpose no specific non-operative measure have yet been shown to improve the outcome in acute in acute pancreatitis. In the surgical management there are various diagnostic, prophylactic and therapeutic option available for both the disease process and its complication but none of them have shown to improve the outcome in acute pancreatitis.

An increased mortality rate associated with the disease is due to inability to asses the severity of the disease at the outset . Various prognostic scoring systems have been developed involving multiple factor and single factor. The drawback with the current severity scoring system is that they are cumbersome and time consuming and lack sensitivity and specificity. In fact there necessity has been questioned10.

Due to change in classification system, lack of statistics in our country and lack of accuracy of scoring system, a better sensitive, specific, severity scoring system which can predict at the outset of the disease is very much needed at present.

This study aims at various complications associated with acute attack of acute pancreatitis and their management.

## AIMS & OBJECTIVES OF THE STUDY

1. To evaluate the various causes acute pancreatitis through proper clinical, biochemical and radiological examination.

2. To study clinical outcome of acute pancreatitis in each etiological factor

## REVIEW OF LITERATURE HISTORICAL

## REVIEW OF ACUTE PANCREATITIS

The earliest description of pancreas dates back to 300BC, given by Herophilus of Chalkaidon. During 100 AD Rufus of Ephesus thought that pancreas acts as a cushion for stomach and named it as " pancreas" meaning "all flesh".

But the earliest description of pancreatitis was available only in 1579 by the French surgeon, Ambrose pares. During the next two centuries, a number of Anecdotal descriptions of pancreatic disease were published. In 1642, Johann Wirsung describe main pancreatic duct and in 1724 GD Santorini describe accessory pancreatic duct which go by their names.

It was only in 1856 that Ancelet prepared a pathological description of acute pancreatitis, pancreatic gangrene and abscess from a review of the reports existing at that time. This gave rise to operative intervention on the pancreas, which was first attempted by Le Dentu 1862.

**Le Dentu**

With the recognition of pancreatitis, various clinical and pathological classification started coming in. First of its kind was by Prince in 1882 who divided acute pancreatitis in to two form an apoplectic and a Sub acute form. This was followed by Reginald Huber Fitz circa, a Boston physician and pathologist. In 1889 he gave clinical description of acute pancreatitis and reported the pathological finding that allowed him to distinguish the hemorrhagic, supportive, and gangrenous form of disease.

**Reginald Huber Fitz**

Nicholas Senn, a Chicago surgeon, suggested operative intervention of acute Pancreatitis associated with pancreatic gangrene or abscess formation in 1886.

**Nicholas Senn**

With the classification and pathological description of pancreatitis, the search for the causative factor began.

In 1901 Opie, at the Johns Hopkins Hospital in Baltimore, documented a gallstone impacted in the ampulla of vater during the postmortem examination of a patient, (operative on by Halsted) who had died of gallstone pancreatitis and there by describe the pathogen tic mechanism of gallstone pancreatitis. It was Synners in 1917 who established alcohol as an important pathological factor.

The importance of pancreas and the severity of its inflammatory disorders were only recognized in 1925 when Berkeley George Andrew Moynihan (lord Moynihan of Leeds) professor of clinical surgery, Leed, England, described acute pancreatitis as "The most terrible of all the calamities that occur in connection with the abdominal viscera". This statement underscores the importance of acute pancreatitis as a major cause of morbidity and mortality today.

In 1929 Elman.R, described the association between elevated serum amylase level and acute pancreatitis. Watts in 1963 described total pancreatectomy for acute pancreatitis.

In 1963 a symposium was held at Marseilles , where pancreatitis was classified on the basis of pancreatitis morphology into Acute pancreatitis, Relapsing panrceatitis, Chronic pancreatitis, and Relapsing

chronic pancreatitis. Lawson in 1970 described early laparotomy with cholecystectomy, gastrostomy and pancreatic drainage in severe acute pancreatitis due to gallstone.

The prognostication of acute pancreatitis was done for the first time in 1974 by John HC Ranson when he was at New York University Medical Centre, New York, Born in Bangalore , India (1938) John Ranson rose to international prominence in medicine in the field of pancreatic disease, and particularly acute pancreatitis. He contributed profound knowledge about nonsurgical and surgical management of acute pancreatitis and his contribution in this field are fundamental to our present understanding of the disease and its clinical management. He was the recipient of many honor and accepted as a leader in the field of acute pancreatitis.

Following Ranson prognostic stratification various stratification systems started coming in. In 1978 from the department of surgery, royal infirmary, Glasgow Clement W Imrie devised a grading system similar to Ranson where only 9 factor needed to be assessed; this system is also well known as Glasgow scoring system. He further modified this system to include only 8 factor also called modified Glasgow scoring system.

Imrie is a very prominent person in the field of acute pancreatitis. He has got various articles published in international

journals to his credit, covering various aspect of acute pancreatitis.

William A Knaus, from the department of anesthesiology, George Washington university medical canter, in the year 1981 developed a system to quantify severity of illness in ICU patient called APACHE system. This system attracted lot of criticism because of its inaccuracies. However it did serve as a prototype for the development of two subsequent system APACHE I and APACHE II.

APACHE II has been widely applied for grading acute pancreatitis. A recent addition is APACHE III system, which is yet to be applied widely to acute pancreatitis.

In the year 1986, Agarwal N from department of surgery, our lady of mercy medical center, Bronx, New York, gave simplified prognostic criteria for acute pancreatitis. He considered fluid requirement and early organ dysfunction as two main adverse criteria for severity. One of these factor, fluid requirement is a factor for evaluation in this study. The other factor, early organ dysfunction has now been included in Atlanta a classification system.

In the field of imaging acute pancreatitis, Emil J Balthazar, Professor of Radiology, Bellevue Medical Center, New York, stands out prominently. In 1989 he gave the CT grading of acute pancreatitis, and emphasized the role of CT in the initial process of diagnosis, as an early predictive indicator of disease severity, and in detecting

complication associated with acute pancreatitis.

Various ill defined terminologies and lot of confusion prevailed with regards to acute pancreatitis. This lead to the symposium at Atlanta where in an universally accepted, clinically based classification system for acute pancreatitis was developed in 1992, all the terminologies related to acute pancreatitis were clearly defined and a sound basis for future studies was established.

In 1993 Linda Rabeneck, physician from the development of internal medicine, Yale university school of medicine, New Haven, constructed a new staging system with clinical and co-morbid features of patient with acute pancreatitis.

Other prominent person who are actively involved and contributed a lot in the field of acute pancreatitis are Hans G Beger, Ulm, Germany, Micheal J McMahan, department of surgery, general infirmary Leeds, England, John P Neoptolemos, department of surgery, Birmingham, England; Edward L Bradley iii, department of surgery, Atlanta, peter Banks, Boston, Buter A.

Indian clinicians who are actively involved in this field are Shisish K Bhansali, Sharad C Shah, Jaslok hospital & Breach Candy hospital Mumbai, India, Sikora S S Department of Surgery, Sanjay Gandhi institute of medical science Lucknow; Chaudhary A; department of surgery; GB pant hospital New Delhi, India.

Laparoscopic surgery is slowly superceding conventional surgery; acute pancreatitis and its complication are amenable for laparoscopic intervention and are practiced successfully by increasing number of Indian surgeon, notable among these being Udwadia, Chowbey, Palanivellu and Thanakumar.

**EMBRYOLOGY OF PANCREAS**

Embryogenesis of pancreas begins during the $4^{th}$ week of gestation. There are nine key processes involved in the development of pancreas. Pancreas develop from two endoderm buds, dossal & ventral, that arise from that part of the gut that later form the second part of the duodenum. The ventral bud arises in close relation to the hepatic bud, in the inferior angle between it and duodenum. The dorsal bud arises from the dorsal aspect of the gut.

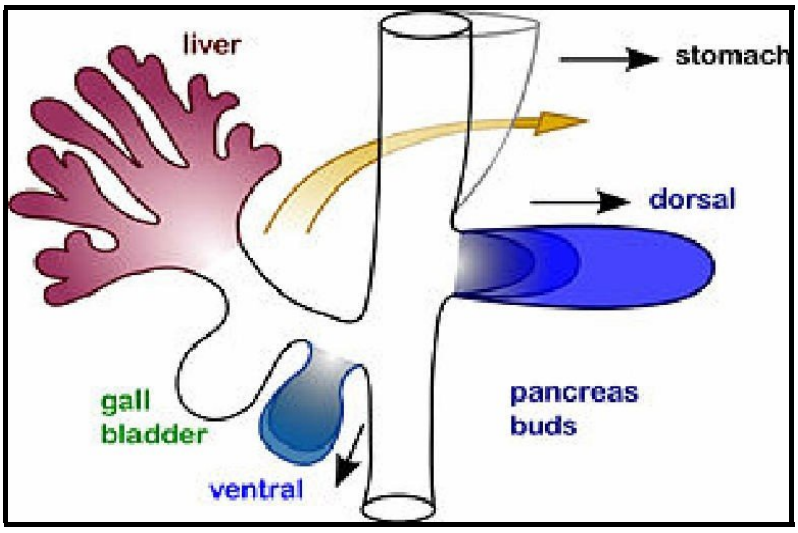



**Fig 1 : Development of PANCREAS - I**

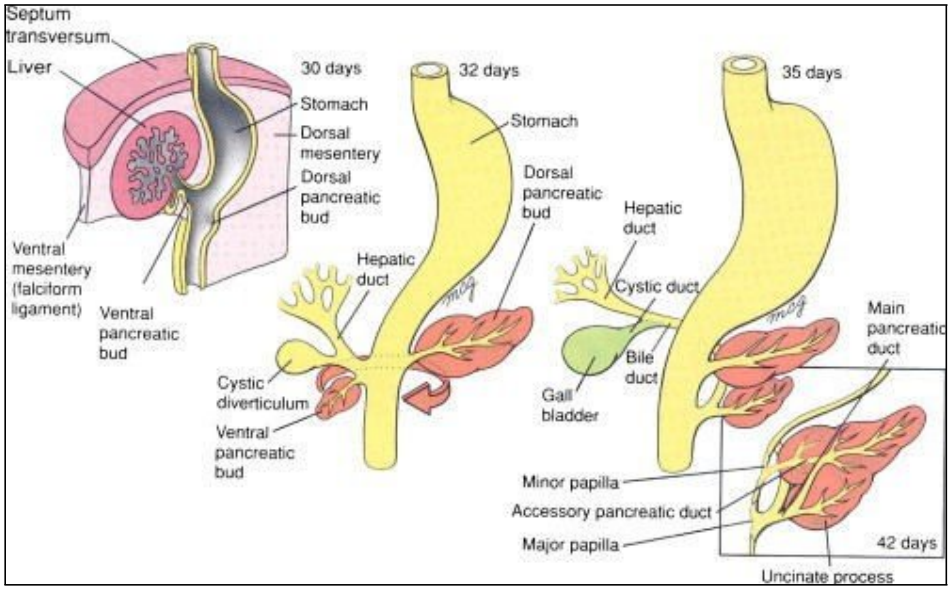



**Fig 2 : Development of PANCREAS - II**

As the duodenum rotates to assume a C- shape configuration, the ventral bud comes to point to the right and dorsal bud to the left. As a result of differential growth of the wall of gut, the attachment of the ventral bud along with bile duct shift to left side.

Pancreatic tissue formed from these two buds now fuses to form one mass. The ventral bud form the lower part of the head, and the uncinate process of the pancreas, while the upper part of the head, the body and tail are formed from the dorsal bud.

Ventral duct and distal portion of dorsal duct form the main pancreatic duct (duct of wirsung) and open in duodenum at the major duodenal papilla , along with the bile duct , proximal dorsal duct form accessory pancreatic duct (duct of santorini) and open in to duodenum through a separate duodenal papilla

The secretary element of pancreas are formed by proliferation of the primitive duct. The islets of Langerhans are also derived from primitive duct system.

**Malformation of pancreatic embryology**

**1-Heterotopic pancreas**

Pancreatic tissue outside the confine of the main gland. Common location are- stomach, duodenum, and small bowel or Meckel diverticulum.

Rare location- umbilicus, colon, appendix, Gall bladder or Omentum. Complication- intestinal obstruction, Ulceration, Hemorrhage.

**Fig 3 : Endoscopic view of heterotropic pancreas in stomach**

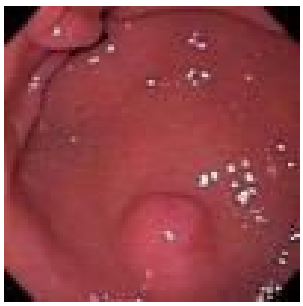
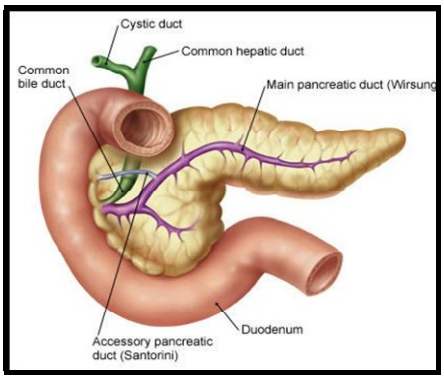


**Fig 4 :-Pancrease Divisum**

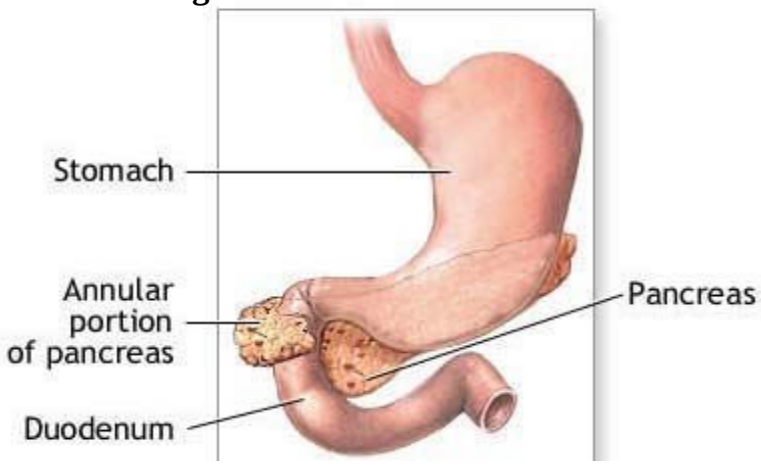



Failure of two primordial ductal systems to fuse. Condition associated with pancreatitis.

**3-Annular Pancreas**

Failure of normal clockwise rotation of ventral pancreatic primordial resulting in normal pancreatic tissue completely or partially encircling the second portion of Duodenum.

**Fig 5 :- Annular Pancreas**



## SURGICAL ANATOMY OF PANRCEAS

The name “pancreas” is derived from the greek “Pan” (all) and “Kreas” (flesh). It was originally thought to act as a cushion for the stomach.

The gland is retroperitoneal lying posterior to the stomach and lesser Omentum. It extends obliquely from the duodenal C- loop to a more cephalad position in the hilum of the spleen. The gland weight from 75 to 125 gm, in length from 10 to 20cm, width from 3 to 5 cm and thickness from 1.5 to 3.5 cm. The gland has a distinctive yellow- tan- pink color and is multilobulated. The pancreas is covered by peritoneum anteriorly and posteriorly it lies in proximity to the inferior vena cava, the right renal vein, the aorta at the level of the $1^{st}$ lumbar vertebra, the superior mesenteric vessel, and the splenic vein. The gland is divided in to 4 portion :the Head, the neck, the body & the tail. The head lies with in the curve of the duodenum overlying the body of the second lumbar vertebra and the vena cava. The portion of pancreas anterior to superior mesenteric and portal vein is designated the neck of the gland. Coming of the side of the pancreatic head and passing to the left and behind the superior mesenteric vein is the uncinate process of pancreas. The body of pancreas lies immediately to left of the neck. The tail of pancreas

extends to the left of the body into the splenic hilum.

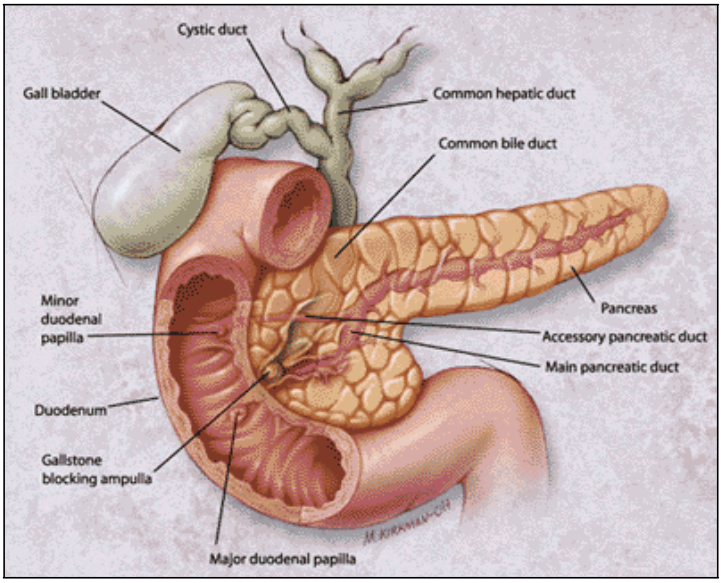



**Fig 6 :-Pancreas showing the adjacent structure**

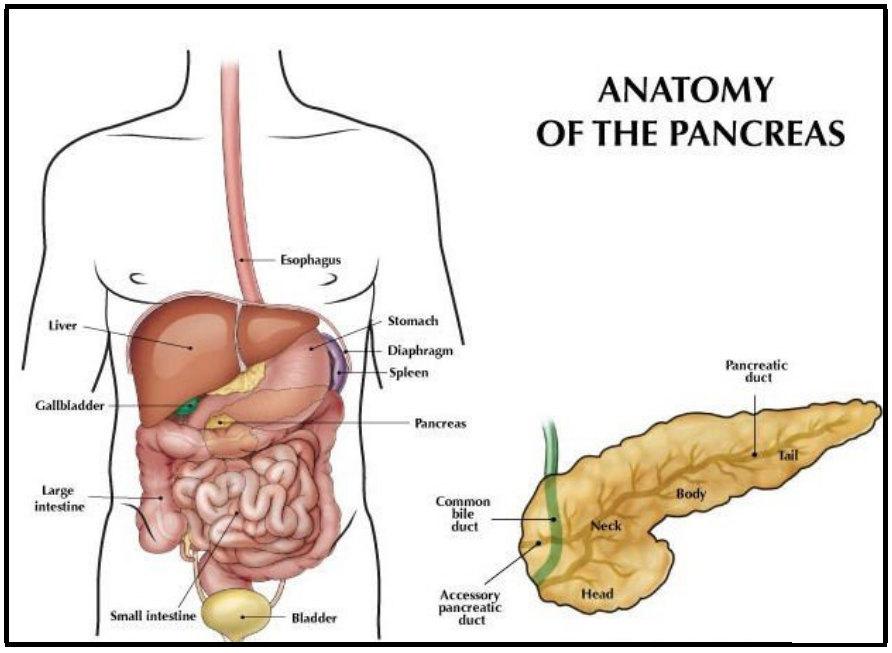



**Fig 7 :-Pancreas and its parts**

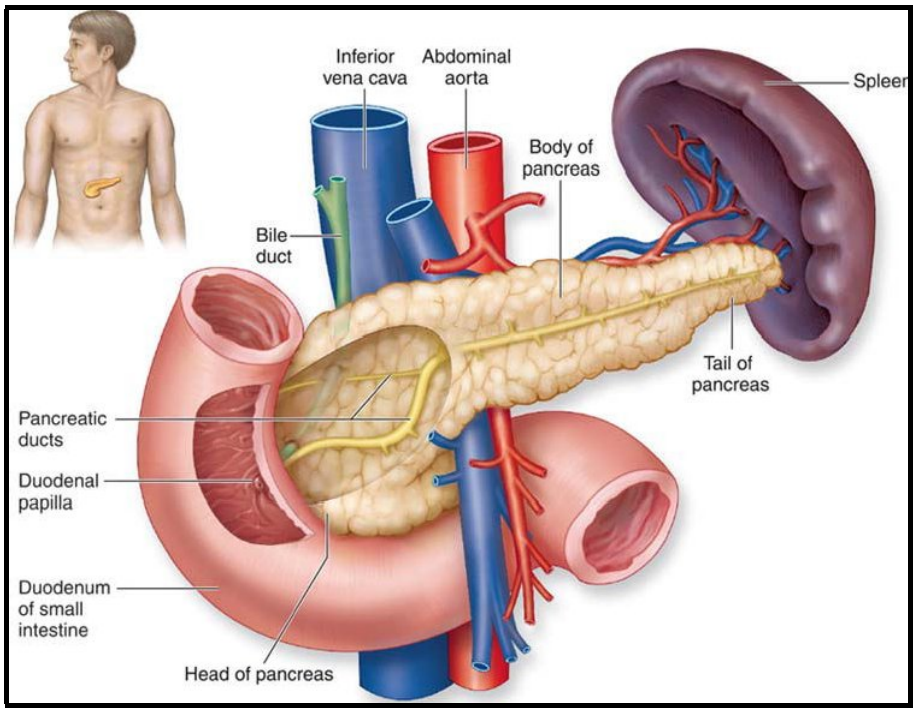



**Fig 8 :-Pancreas showing surgical important structure**

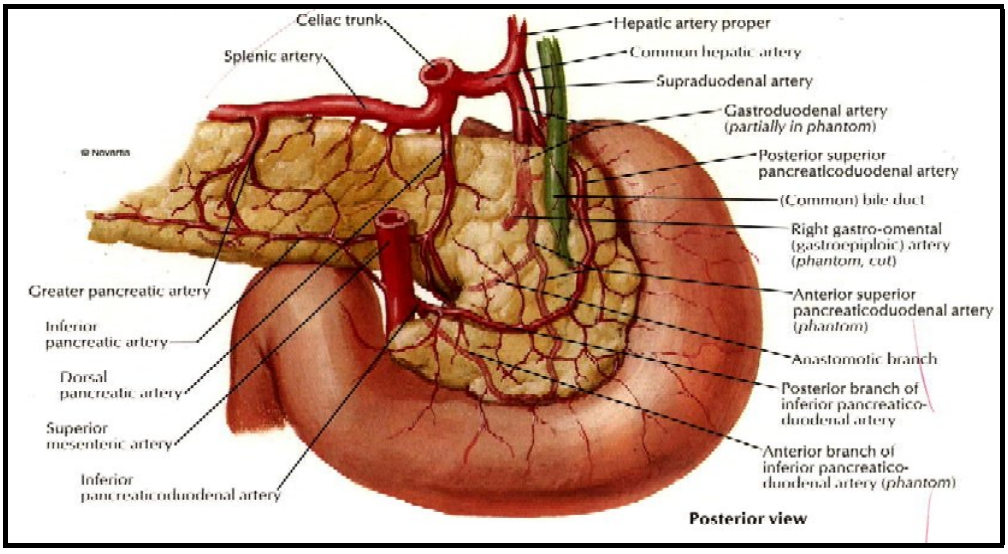



**Fig 9 :-Arterial supply of pancreases**

An extensive arterial system originating from multiple sources supplies the pancreas. Head of the pancreas is supplied by the anterior & posterior pancreatico-duodenal arteries, which in turn are branches of superior & inferior pancreatoco-duodenal arteries arising from celiac axis and superior mesenteric artery respectively.

The blood supply to the body and tail of the pancreas is through splenic and gastroepiploic arteries. Venous drainage corresponds with the arterial anatomy.

Multiple lymph node groups drain the pancreas. Head is drained by, node in pancreatico-duodenal groove, subpyloric, portal, mesocolic, mesenteric and aortocaval node. Body and tail of pancreas drain to retro peritoneal nodes in the splenic hilum or to celiac, aortocaval, mesocolic, or mesenteric nodes.

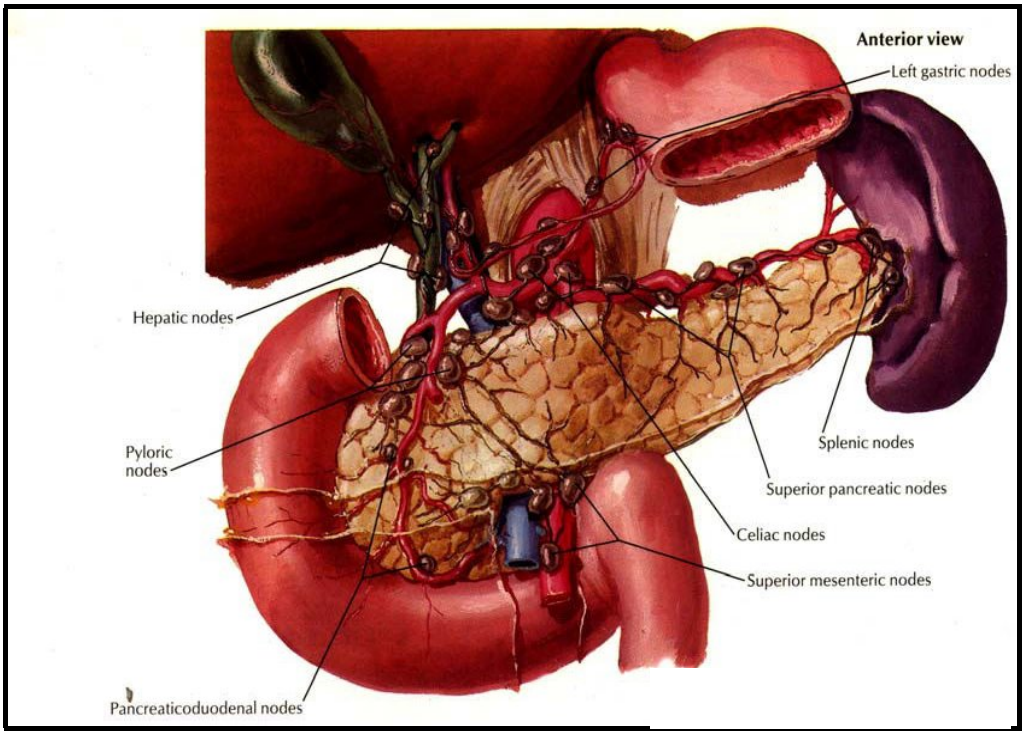



**Fig 10 :-Lymphatic drainage of pancreas**

Pancreas has both sympathetic & parasympathetic innervations immediately to the left of the neck, the tail of the pancreas extend to the left of the body into the splenic hilum.

## HISTOLOGY

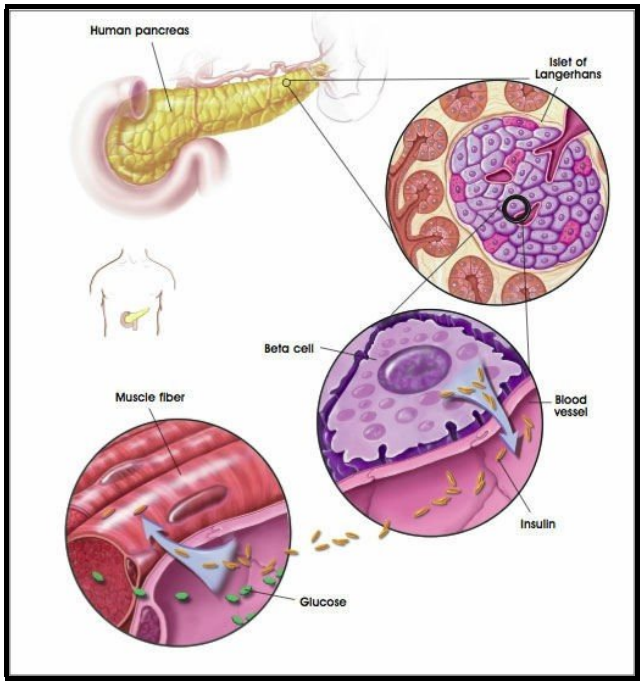
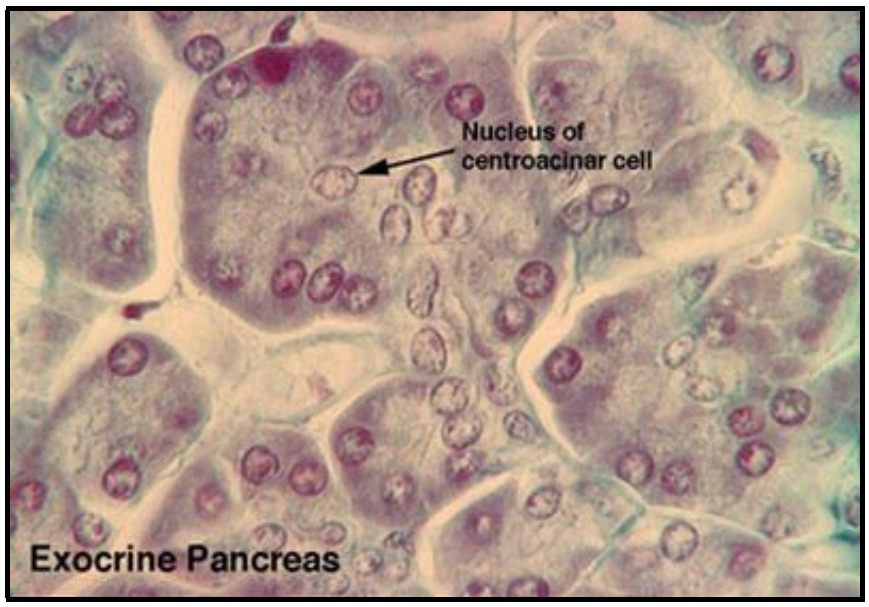
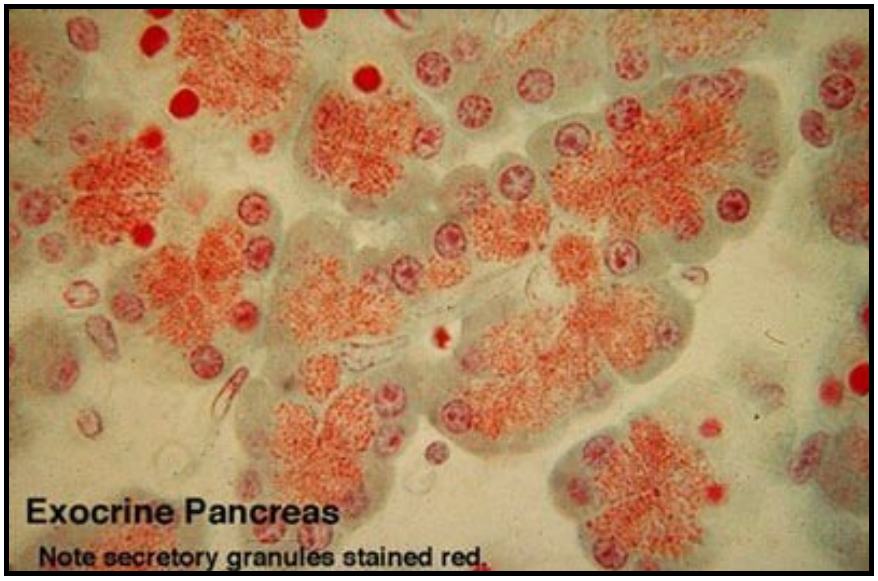



**Fig 11 :-Endocrine system of pancreas**

Two distinct organ system share residence with the human pancreas, the endocrine system and the exocrine system. The acini and ductal system constitute the exocrine portion of pancreas. Acinar cell contain zymozen granules. Acinar cell constitute about 80% of pancreatic mass. Pancreatic acinar tissue is organized into lobules. The islets of langerhans are distributed throughout the pancreas. Islet cells consist of differing cell type. 75% are B cell (producing insulin), 20% A cells (producing glucagon) and the remaining are D cells (producing somatostatin)

**Fig 12(A & B) :-MICROSCOPIC VIEW OF PANCREAS**



## PHYSIOLOGY OF PANCREAS[11]

Pancreas plays a vital role in digestion of food in the gut and in glucose homeostasis. Pancreas has exocrine and endocrine secretion. Exocrine secretion has two distinct components, enzyme secretion originating from acinar cells and electrolyte secretion originating from the centriacinar and intercalated duct cells

- ✓ pH-8
- ✓ Specific gravity-1.007 to 1.035
- ✓ Basal secretory- 0.2 to 0.3 ml / min
- ✓ Enzyme secretion- 6 to 20 mg/day
- ✓ Bicarbonate rich fluid- 2.5ltrs/day

**Enzyme secretion**

The pancreatic juice contains enzymes that are of major importance in digestion. Its secretion is controlled in part by a reflex mechanism and in part by gastrointestinal hormone, secretin and CCK.

**TABLE 1 : PRINCIPAL DIGESTIYE ENZYMES**

| ENZYMES | ACTIVATORS | SUBSTRATE |
|---|---|---|
| Trypsin | Enteropeptidase | Protein &polypeptide |
| Chymotrypsin | Trypsin | Protein& polypeptide |
| Elastase | Trypsin | Elastin |
| Carboxipeptidase A | Trypsin | Protein & polypeptides |
| Carboxipeptidase B | Trypsin | Protein& polypeptides |
| Colipase | Trypsin | Fat droplets |
| Pancreatic lipase | ……. | Triglycerides |
| Cholesteryl ester hydrolase | …….. | Cholesteeyl ester |
| Pancreatic amylase | Cl - | Starch |
| Ribonuclease | ……. | RNA |
| Deoxyribonuclease | ……. | DNA |
| Phospholipase A2 | trypsin | phospholipid |

Powerful protein spliting enzymes of the pancreatic juice are secreted as inactive proenzymes. Trypsinogen is converted to the active enzymes trypsin by the brush border enzyme enteropeptodase when the pancreatic juice enter in to duodenum.

Trypsin convert chymotrypsinogens into chymotrypsin and other proenzyme into enzymes. Trypsin can also active Trypsinogen, therefore once some trysin is formed, there is an autocatalytic chain reaction.

The potential danger of the release into pancreas of a small amount of Trypsin is apparent, the resulting chain reaction would produce active enzyme that could digest the pancreas. It is therefore not surprising that the pancreas normally contains a Trypsin inhibitor.

Another enzyme activated by trypsin is phospholipase A2. This enzyme split a fatty acid of lecithin, forming lysolecithin. Lysolecithin damages cell membrane. It has been hypothesized that in acute pancreatitis phospholipase A2 is activated in pancreatic duct, with the formation of lysolecithin from the lecithin that is a normal constituent of bile. This cause disruption of pancreatic tissue and necrosis of surrounding fat.

Small amount of pancreatic digestive enzymes normally leak into the circulation, but in acute pancreatitis the circulating level of the digestive enzyme rise markedly. Measurement of plasma amylase or lipase concentration is therefore of value in diagnosing the disease.

**ETIOLOGY**

Alcohol intake and biliary tract disease account for majority of the cases (90%) Relative frequency depends on the patient population and prevalence of alcoholism in the population studied. In united states alcohol abuse is the main cause. In Europe and Asia, gall stone

associated pancreatitis predominates. Also females are more prone to gall stone pancreatitis and males for alcohol induced pancreatitis.

## CAUSES OF ACUTE PANCREATITIS

### OBSTRUCTION

- Choledocholithiasis
- Ampullary or pancreatic tumour
- Worms or foreign bodies obstructing the papilla
- Pancreas divism with accessory duct obstruction
- Choledochocele
- Periampullary duodenal diverticula
- Hypertensive sphincter of Oddi

## TOXIN OR DRUGS

**TOXIN-**Ethylalcohol,methylalcohol, scorpion venom, organophosphorus insecticides.

**Drugs -** Azathiaprine and Mercaptopurin, Valproic acid, Estrogens, Tetracycline, Metronidozole, Nitrofurantoin, Pentamidine, Furosemide, Sulfonamide, Methyldopa, Cimetidine, Ranitidine, Sulindac, Didanosine, Acetaminophen, erythromycin, salicylates.

## TRAUMA

Accidental - Blunt trauma to the abdomen

Iatrogenic - postoperative trauma, ERCP, Endoscopic sphincterotomy

## METABOLIC ABNORMALITIES

- Hyper triglyceridemia
- Hypercalcemia

**INHERITED CONDITION**

**INFESTION** -

Parasitic- Ascariasis, Clonorchiasis

Viral - Mumps, Rubella, Hepatitis A,B, non-A,non-B, Coxsackievirus B, Echo virus, adenovirus, cytomegalovirus, varicella, Epsteinbarr virus, Human Immunodeficiency virus.

Bacterial- Mycoplasma, Campylobacter jejuni, Mycobacterium tuberculosis, M, avium complex, Legionella, Leptospirosis.

**VASCULAR ABNORMALITIES**

ISCHEMIA - Hypoperfusion
Atherosclerotic
emboli

VASCULITIS - SLE, PAN, Malignant hypertension.

## MISCELLANEOUS CONDITIONS

- ✓ Penetrating peptic ulcer
- ✓ Crohn's disease
- ✓ Reye' syndrome, Cystic fibrosis
- ✓ Hypothermia

## IDIOPATHIC CAUSE

## ETIOPATHOGENESIS

### *GALL STONE PANCREATITIS*

The etiology of gallstone in the pathogenesis of pancreatitis was first suggested by Opie[2]. He proposed that acute pancreatitis was initiated by a stone impacted in the ampulla of vater which resulted in the diversion, producing reflex of bile into the pancreatic duct. Pancreatic injury can be initiated by the migration of stone and not necessarily by the impaction of stone.

Additional evidence to support the gallstone migration theory comes from cholangiographic studied that demonstrate a common channel for CBD and pancreatic duct in up to 90% of pancreas with a history of gall stone pancreatitis, compared with only a 20-30% incidence of a common channel in patient with calculus biliary tract disease and no history of pancreatitis[12]. Recent experimental evidence shows that biliary pancreatic reflux is not essential for initiation of gall stone pancreatitis[13].

**ALCOHOL**

There is undoubtedly an association between alcohol abuse and acute pancreatis although the exact mechanism of alcohol related injury is unknown. Several theories exist

1. Pancreatic parenchyma is injured by pancreatic enzyme extravasation, facilitated by an increase in pancreatic ductal permeability, in the face of exocrine hypersecretion and partial ampullary obstruction[14].
2. Protein pluging of pancreatic duct caused by alcohol may initiated enzyme extravasation with resultant injury

3. Transient state of hypertriglyceridemia induced by alcohol ingestion cause toxic level of free fatty acid produced from lipolysis of triglycerides which may induced pancreatic injury by causing acinar cell or capillary endothelial cell injury.
4. Toxicity from acetaldehyde the first stable product of ethanol Oxidation[15].

**HYPERLIPIDEMIAS**

Hyperlipidemias alone without alcohol abuse can cause acute pancreatitis. Primary hyperlipidemias- Ferdrickson's type I and type V notable for hypertriglyceridemia and chylomicronemia. Secondary hyperlipidemias- extraneous estrogen administration, nephritis, and castration may also be the cause of acute pancreatitis.

**HYPERCALCEMIA**

Hypercalcemia due to hyperparathyroidism cause acute pancreatitis. Mechanism involve are:

1. Calcium stimulate pancreatic hypersecretion
2. Calcium induced trypsinogen activation with subsequent parenchymal auto destruction
3. Calcium associated stone precipitation in the pancreatic duct causing ductal
4. Obstruction.

## IDIOPATHIC PANCREATITIS

Recent studies have clarified the etiology of acute pancreatitis in many patient once classified as having idiopathic pancreatitis. 60% of these patient were identified to have biliary sludge termed microlithiasis[16,17] (suspension of cholesterol monohydrate crystal or calcium bilirubinate granules). cholecystectomy or endoscopic sphincterotomy prevented release of pancreatitis in these patients.

## PATHOLOGY[18]

## MORPHOLOGY

The morphology of acute pancreatitis necrosis stems directly from the action of activated pancreatic enzymes that are released into the pancreatic substance. The basic alternation are :-

1. Proteolytic destruction of pancreatic substance,
2. Necrosis of blood vessels with subsequent hemorrhage
3. Necrosis of fat
4. An accompanying inflammatory reaction. The extent and predominance of each of these features depend on the duration and severity of process. In the very early stages, only interstitial edema is present. Soon after, focal and confluent area of frank necrosis of endocrine and exocrine tissue are found.

The peritoneal cavity contains a serous and slightly turbid fluid in which globules of oil can be identified. Foci of fat necrosis may be found in any of fat depots

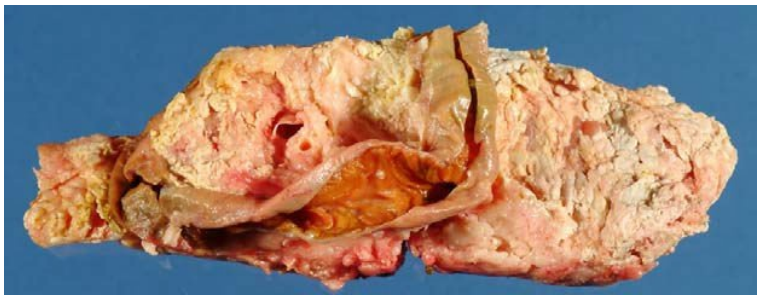


**Fig 13 :- Pancreas with the inflammed and necrotic area**

## HISTOPATHOLOGY

Focal areas of fat necrosis occur in pancreatic and peripancreatic fat. Following enzyme destruction, adipocytes are transformed into shadowy out lines of cell membranes filled with pink, granular opaque precipitates amorphous basophilic calcium precipitates may be visible with in the necrotic focus. Neutrophillic infiltration and interstitial hemorrhage eventually ensure

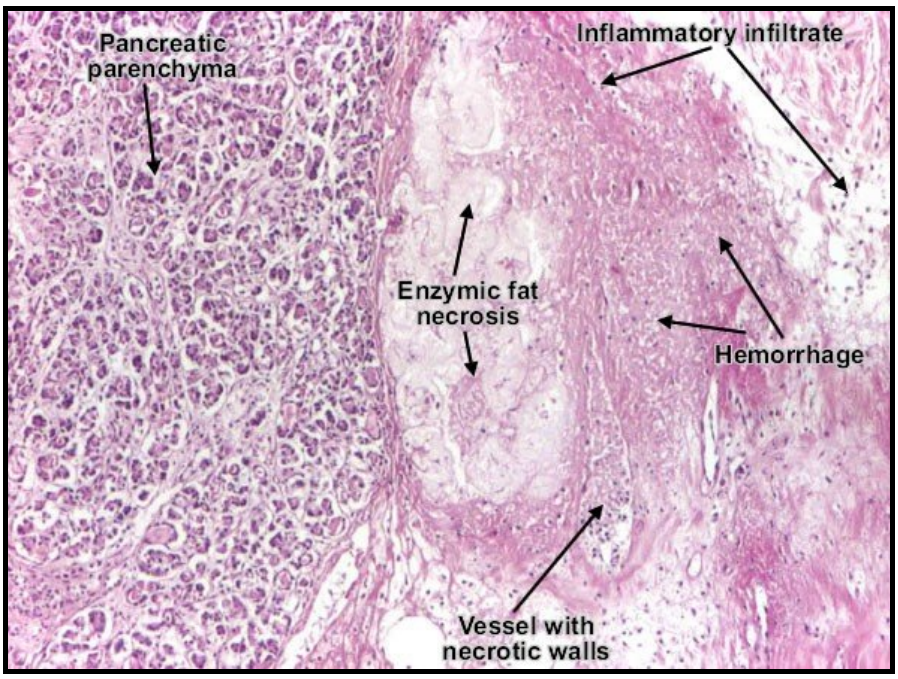



**Fig 14 :-Histopathology of acute pancreatitis**

## PATHOPHYSIOLOGY OF ACUTE PANCREATITIS

Owing to relative inaccessibility of the human pancreas and complexity of the disease most of the pathophysiological data on the mechanism of pancreatic inflammation and necrosis are based on animal model. Experimental studies in rats using cholene deficient diet and cerulin infusion suggest that these pancreatitis inducing substance cause a blockade in the secretion of zymogen granules from individual acinar cells which results in fusion of zymogen granules with intracellular proenzyme Trypsinogen yielding active intracellular trypsin which is capable of cellular auto-digestion[19].

Finding of acinar cell zymogen granule enlargement and formation of large Autophagosomes have been found in human tissues in acute pancreatitis

## PATHOPHYSIOLOGY OF REGIONAL NECROSIS

Three key factor, acting sequentially account for regional necrosis

1. Inrtaglandular activation of pancreatic enzymes cause pancreatic enzyme cause auto digestion.
2. Over stimulation of inflammatoryeffectors cells like macrophages, polymorphs.
3. Vascular mechanism- ischemia, reperfusion, hemorrhage.

Intra-acinar activation of proteolytic/lipolytic pancreatic enzymes particularly trypsin seems to be the initial mechanism that triggers auto digestion.

Once trypsin is generated in excess, it exhausts pancreatic secretory trypsin inhibitors in the acinar cell and non- specific antiprotease [alfa2 – macroglobulin] in the interstitium. Free trypsin then activates other zymogen and several cascade system of protease[ complement system, kallikreun- kinin, coagulation and fibrinolysis]

Activation of complement system by trypsin and subsequent generation of C5a play a major role in the recruitment of inflammatory cells. Over stimulation of macrophages and neutrophils results in the local spilling of arachidonic acid metabolites, proteolytic and lipolytic enzymes, clot promoting factors, platelet-activating factor and cytokines, which exceed the scavenging capacity of endogenous antioxidant systems. These substances interact with pancreatic microcirculation to produce thrombosis and hemorrhage.

Local circulatory effects play a significant role in the extension and complication of pancreatic necrosis. Endothelial cell damage and local activation of the coagulation system by trypsin results in interstitial edema and fibrin- platelet thrombus formation in the pancreatic microcirculation. The subsequent pancreatic ischemia result in reduction in

local and regional input if antiproteases and amplifies the premature activation of trypsinogen.

This cascade of multiple activation of enzymes leads to the rapid exacerbation of the inflammatory process and to the gradual widening of regional necrosis.

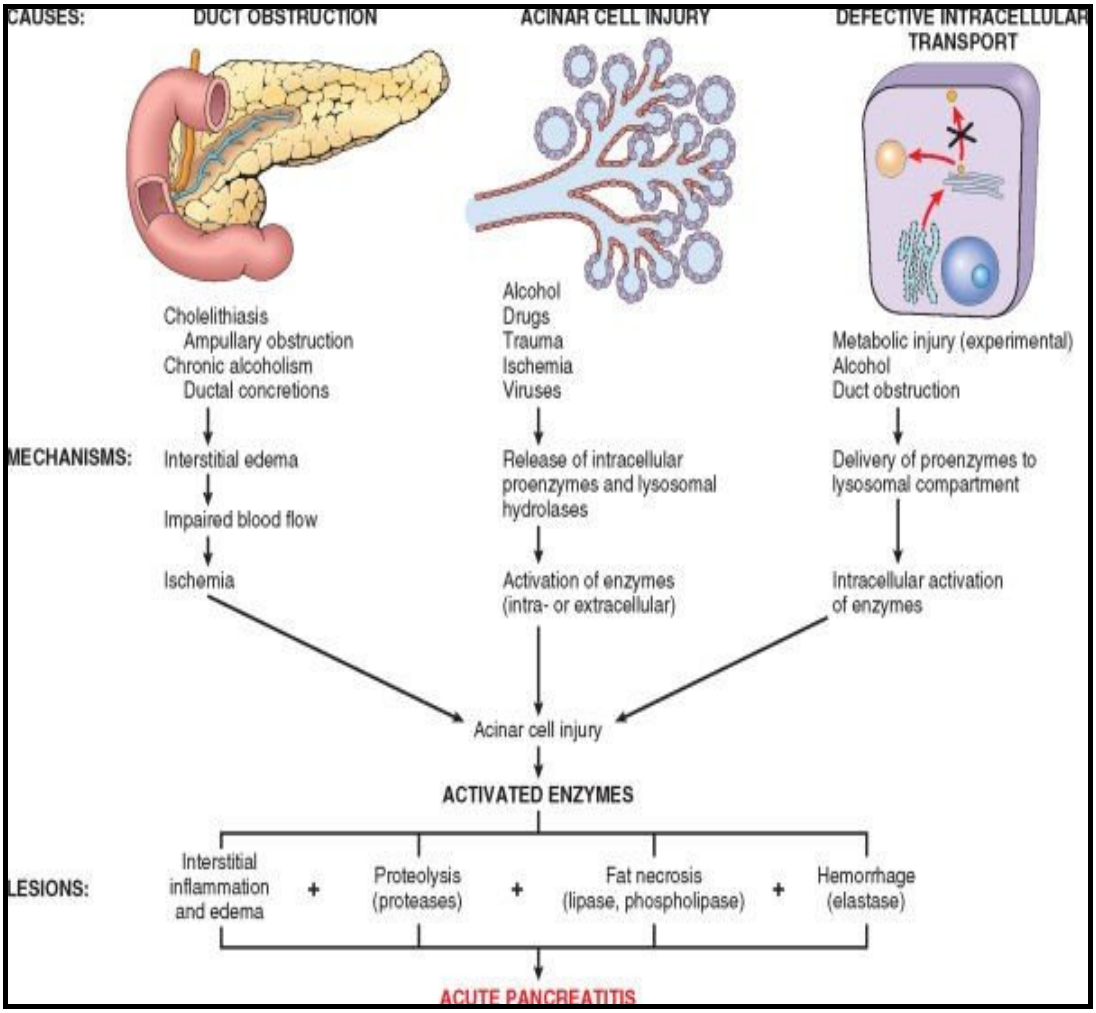



**Fig 15 :- Pathophysiology of acute pancreatitis**

## PAHTOPHYSIOLOGY OF REMOTE ORGAN DYSFUNCTION

The clinical course of acute pancreatitis can be divided into two successive phase.

1. The early 'Toxemic' phase (0-15 days) characterized by distant organ damage leading to multi organ failure.
2. The late 'Necrotic' phase [after 2nd week] characterized by locoregional complications.

The systemic complication that characterize the early stage of the attack, particularly cardiocirculatory and respiratory failure, are mainly due to spilling of active pancreatic enzyme and toxic substance from the retro peritoneum into systemiccirculation. The route of autointoxication is mainly through retroperitoneal and peripancreatic lymphatics, which drain into the thoracic duct.

The homodynamic profile of early phase is characterized by low systemic vascular resistance, increased cardiac output, oxygen uptake, pulmonary shunt fraction and severe myocardial depression. These changes are attributed to

1. Intravascular volume depletion because of increased vascular permeability, abnormal fluid sequestration and gastrointestinal or retro peritoneal hemorrhage.

2. Direct or trypsin mediated activation of contact system
3. Release of myocardial depressant factor by acinar cells
4. Release of prostanoids and cytokines notably tumor necrosis factor by activated leucocytes.

The homodynamic response is similar to gram-negative sepsis, which includes increased resting energy expenditure, elevated protein catabolism, hepatic gluconeogenesis and peripheral insulin resistance.

Respiratory complication arises from combination of pleural effusion, elevated diaphragm to pain/ ileus, atelectasis, hypoxemia and pulmonary infection. About 20% suffer from early ARDS, the cause being damage and subsequent increased permeability of alveolar capillary membrane. Other mediators of ARDS are pulmonary leucostasis, proteolytic enzymes [elastase]. Phospolipase A2 hypertriglyceridemia and activated coagulation system by trypsin. Other causes of respiratory failure are pulmonary embolism, aspiration and nosocomial pneumonia.

Pleural effusion found in acute pancreatitis are due to transfer of peripancreatic exudates via the peri- diaphragmatic lymphatic plexus

DIC results from photolytic effects of circulation trypsin. Oliguria and renal failure are due to hypovolemia, deposition of fibrin in the glomerular capillaries and acute tubular necrosis.

## PATHOPHYSIOLOGY OF LOCAL COMPLICATION

Pancreatic phlegmon refers to edematous, inflammatory and necrotic sterile tissue in and around the pancreas. The necrotic tissue gets infected (infected peripancreatic collection) in about 40% of patient, more so in gall stone pancreatitis. The risk of pancreatic sepsis is maximum in the third week. Early infection is mostly by a single gram negative organism GIT origin by translocation, perforation, infected bile or post ERCP or via hematogenous or lymphatic spread. Infected pancreatic necrosis in which necrotic tissue predominates present early and has higher mortality.

Pancreatic abscess consists of pus enclosed by inflammatory walls resulting from infection of liquefied necrotic areas. Pancreatic abscess present after the active phase is over and run a more indolent course with fewer complications.

Pancreatic pseudocyst develops in 10-20% of patient and is more common in alcoholic pancreatic complication, though sepsis and hemorrhage are more likely in cysts associated with gallstone. Acute pseudocyst consist of an effusion of pancreatic Juice rich in amylase that lacks in epithelial lining and has become gradually enclosed by

fibrosis walls after a period of 4-6 weeks[21]. Half of the pseudo cyst resolve spontaneous in 1-2 month, some of them fistulise into the peritoneal cavity ( pancreatic ascites) or into chest( pancreatico- pleural or pericardial fistula) where secondary infection or bleeding can occur.

Gastrointestinal and retroperitoneal hemorrhage can occur due to gastric ulcers, esophageal varices, erosion of visceral vessels and pseudo aneurysm.

Coloniccomplication include mechanical obstruction, rectal bleeding, and colonic necrosis with perforation and fistula formation. Colonic damage is attributed to direct toxic effect of pancreatic enzymes (pericolitis) or colonic vascular ischemia (due to DIC, thrombosis of superior mesenteric vein, hypotension)

## CLINICAL PRESENTATION OF ACUTE PANCREATITIS

A reasonably secure diagnosis is often possible based solely on clinical presentation. In more than 90% of patients the predominant features is pain abdomen. Pain is mostly in the epigastrium, constant, boring or penetrating and may radiate to the back. Pain start 12-48 hour a bout of alcohol or after a large meal in case of gallstone pancreatitis. Fever, vomiting, tachycardia, epigastric tenderness, hypoactive and absence bowel sound, and abdominal distension are other feature. Some times a mass can be palpable in the epigastrium. In about 3% patient, hemorrhagic panceatitis can cause bluish discoloration in the left flank (Grey Turner' sign) or around the umbilicus(Cullen's sign). These signs, are due to tracking of blood stained retroperitoneal fluid planes of abdominal wall to the flank or along the falciform ligament to the umbilical area, and indicate severe episode of acute pancreatitis[22]. Jaundice may be present and represent distal common bile duct obstruction by a gallstone or compression by pancreatic edema. In severe acute pancreatitis hypovolemia, hypotension, hypo perfusion and obtundation may be present

Other manifestations include pleural effusion (mostly left side), ascites, and elevation of left hemi- diaphragm with tachypnoea, dyspnoea and cynosis. Less commonly subcutaneous fat necrosis,

cerebral abnormalities of a non- lateralizing nature and disseminated intravascular coagulation can be seen.

**DIAGNOSTIC WORK UP**

The direct inspection of pancreas at laparotomy with microscopic examination of pancreatic tissue is only way to confirm the diagnosis of the acute pancreatits. In routine clinical practice, clinical feature particularly pain abdomen, nausea/ vomiting and raised serum amylase are the diagnostic cornerstone. The elevation of serum amylase (normal 60-180U/L) is observed within 24 hours of the onset of symptoms and gradually return to normal in the subsequent weeks. Persitent elevated serum amylase beyond initial week of illness reflect ongoing pancreatic inflammation or development of pancreatic complication, pseudocyst, phlegmon or abscess. Serum amylase determination has high sensitivity(>95%[8,23]) but over all specificity is low (70%), since elevated serum level occur in many condition( intra abdominal and extra abdominal).

Furthermore, amylase level is not raised in 5% of cases being hyperlipidemic pancreatitis, extensive pancreatic necrosis, and chronically diseased pancreas.

Improved accuracy in diagnosis of acute pancreatic can be achieved by measuring amylase isoenzyme component. P-type

isoenzyme, which arises from pancreas account for 40% total circulatory amylase, is raised out of proportion in acute pancreatitis, peptic ulcer perforation, bowel obstruction and mesenteric infarction. S-type isoenzyme is seen in ruptured ectopic pregnancy, salivary gland disorder and salphingitis etc.

**TABLE 2: DISORDERS ASSOCIATED WITH HYPERAMYLASEMIE**

| INTRA-ABDOMINAL | EXTRA-ABDOMINAL |
|---|---|
| *Pancreatic disorders*<br>Acute pancreatitis<br>Chronic pancreatitis<br>Trauma<br>Carcinoma<br>Pseudocyst<br>Pancreatic ascites Abscess<br><br>*Non pancreatic disorders* Biliary tract disease Intestinal obstruction<br>Mesenteric infarction<br>Perforated peptic ulcer Peritonitis<br>Afferent loop syndrome Acute | *Salivary gland disorders*<br>Mumps<br>Parotitis<br>Trauma<br>Carcinoma<br>Calculi<br>Impaired amylase excretion Renal failure<br>Macroamylasemia<br><br>*Miscellaneous*<br>Pancreatic pleural effusion Pneumonia<br>Mediastinal pseudocyst<br>DKE (Diabetic Keto acidosis) Pregnancy |

Urinary amylase excretion (normal 4-400 U/L) is more sensitive index of acute pancreatitis though not diagnostic.

Amylase–creatinine clearance ratio has been used in the diagnosis of acute pancreatitis. This is calculated by the equation.

***Urine amylase/serum amylase X serum creatinine/urine creatinine***

The ratio varies from 1-5%. A ratio more than 6% is consistant with the diagnosis of acute pancreatitis. This is also disfavoured because the amylase creatinine clearance ratio may be raised in diabetic ketoacidosis, burns, renal insufficiency, perforated peptic ulcer, pancreatic carcinoma, etc.

Serum lipase elevation is a more specific indicator of acute pancreatitis than serum amylase because lipase circulating in the serum is mostly of pancreatic Origin[8] Lipase is elevated for longer period and hence useful in patient who present late. But, serum lipase is not most specific for acute pancreatitis, as it can be raised in perforated peptic ulcer, acute cholecystitis and intestinal ischemia.

Diagnostic paracentesis and analysis of peritoneal fluid for elevated amylase and lipase combined with serum elevation of the same has been strongly correlate with acute pancreatitis. Serum latescence is another indicator of acute pancreatitis due to hyperlipidemia and alcohol. Methemalbumenemia has been suggested

as a marker for acute hemorrhagic pancreatitis but it lacks sensitivity or specificity.

Hemoconcentration, leucocytosis, hyperglycemia, hypocalcemia, mild azotemia hyperbilirubinemia, elevation of ALT, ALP and GTT, coagulation abnormalities marked by hyper coagulability, hypofibrinogenemia and DIC are other hematological changes seen in acute pancreatitis.

**RADIOLOGICAL PROCEDURE**

Plain radiograph of the abdomen may reveal paralytic ileus, increased gastro colic separation, sentinel loop ( dilated proximal jejunal), colon cut-off sign( distension of colon at the level of transverse colon with no gas in splenic flexure); cholelithiasis, obliteration of psoas margins[23].

Plane radiograph also rules out potential abdominal emergencies like perforation of hollow viscous or mesenteric ischemia.

A chest radiograph may show left pleural effusion, elevated left hemi diaphragm, basal atelectasis and also delineates other causes of plain abdomen like left lower lobe pneumonia or pneumoperitoneum. In multiorgan failure if lung is affected ARDS changes are seen on chest X –ray.

Upper GI contrast studies may show widening of 'C' loop of duodenum, anterior displacement of stomach and duodenal mucosal abnormalities, but are not longer favored as these finding are not specific.

Abdominal ultrasound examination cab be inconclusive and often misleading. It is largely operator dependant and in 30-40% of patient's pancreas cannot be visualized due to air filled bowel loops. It is the also inaccurate in detecting pancreatic necrosis and regional inflection. Still, it can be used to detect pancreatic edema, peripancreatic fluid collection, gallstone causing pancreatitis, biliary sludge and also pseudocyst, ascites, portal or splenic vein thrombosis.

CT scan is currently the most sensitive non invasive method to confirm the diagnosis for acute pancreatitis[26]. The specificity of an admission CT scan is found 100% and sensitivity is 85%. Most episodes undetected by CT scan are mild and CT scan also provides alternate diagnosis in case with false positive elevation of enzymes. Also contrast enhancement differentiates between edematous and necrotizing pancreatitis.

**CT FINDING IN ACUTE PANCREATITIS**

**PANCREATIC CHANGES**

- ✓ Parenchymal enlargement-diffuse, focal
- ✓ Parenchymal edema
- ✓ Necrosis

**PERIPANCREATIC CHANGES**;

- ✓ Blurring of fat planes
- ✓ Thickening of fascial planes
- ✓ Presence of fluid collection

**NON- SPECIFIC SIGNS**

- Pleural effusion

- Bowel distention

- Mesenteric edema

CT scan is also useful in demonstrating structural complication that develop during the course of acute pancreatitis like pancreatic abscess, pseudocyst or fluid collection. Also severity of acute pancreatitis can be graded using CT scan and has been used in prediction of prognosis [9,27,28,29].

MRI provides a more accurate diagnosis of acute pancreatitis and equivalent information, as CT scan, about extent of fluid collection and parenchymal irregularities.

ERCP should be done only in patient with acute pancreatitis who clinical, course fail to improve despite full intensive care support and in whom ampullary or common bile duct stone impaction is suspected based on ultrasound or clinical/ biochemical sign of cholangitis. Risk of exacerbation of pancreatitis, introducing infection into devitalized pancreatic area, failure of pancreatic catheterization due to duodenal stenosis by pancreatic swelling and papillary edema limit the use of ERCP in the early stages of pancreatitis.

However , ERCP may be more useful in patient who have recurrent attacks of acute pancreatitis without an obvious etiology. It is useful in identifying potentially correctable lesion such as CBD stones with impaction, pancreas divisum, ampullary stenosis etc where appropriate intervention can prevent recurrent pancreatitis in more than 80% of patient.

Studied have shown that early endoscopic sphincterotomy improves prognosis in patient with gall stone pancreatitis with jaundice and cholangitis[30,31,32,33,34,35]

**Fig 16(a):- USG showing Acute Pancreatitis**

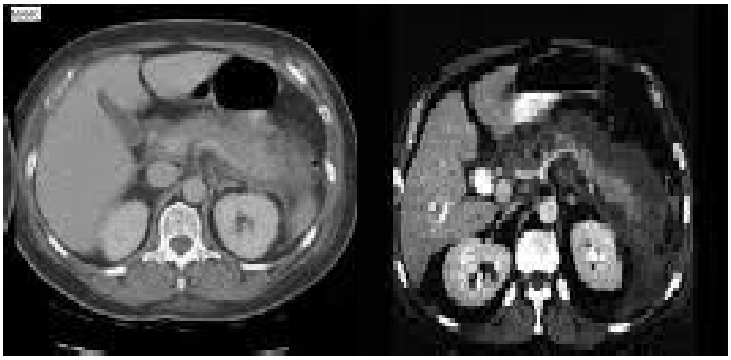


**Fig 16(b):- CECT showing Acute Pancreatitis**

## PROGNOSTIC STRATIFICATION

Early assessment and prediction of severity are of outstanding importance to avoid costly and invasive monitoring and treating in the largest group of patient, who tend to turn a benign course. Accurate prediction of severity and monitoring are necessary in severe form in an intensive care unit to anticipate early and complication to consider aggressive treatment and to judge the response to therapy

## MULTIFACTOR SCORING SYSTEM

One of the early system for judging severity was developed by Ranson in 1936. it incorporates five feature measured at admission and six additional criteria Determined during the initial 48 hours. The criteria were refined to create two similar systems, one for alcoholic

pancreatitis and for gallstone pancreatitis. Patient with zero to two Ranson prognostic sign have essentially no mortality and do not require anything more than simple supportive care. Patient with three or four signs have a mortality of 15% and 40% of these patient require intensive care therapy.

Patient with five or six signs have a mortality rate of approximately 50% and almost universally require support in an intensive care unit. Patient with seven or more prognostic signs have a predicted mortality of almost 100%.

**RANSON'S CRITERIA[36]**

**ALCOHOLIC PANCREATITIS**

***on admission to hospital***

- ✓ Age >55 years
- ✓ White blood count> 16000/$mm^3$
- ✓ Glucose> 200mg/dl
- ✓ Lactate dehydrogenage > 350 U/L

- ✓ Aspartate aminotransferase>250 U/L

***within 48 hours of* admission**

- ✓ Decrease in hemotocrit >10 %
- ✓ Increase in blood urea nitrogen > 5 mg/ dl
- ✓ Serum calcium < 8 mg / dl
- ✓ Arterial oxygen pressure< 60mm Hg
- ✓ Base deficit >4mmol/Ltr
- ✓ Fluid sequestration> 6Ltr
- ✓

**GALLSTONE PANCREATITIS**

***On admission to hospital***

- ✓ Age >70 year

- ✓ White blood count>18,000
- ✓ Glucose >220 mg/dl
- ✓ Lactate dehydrogenase>400U/L
- ✓ Aspartate aminotransferase>250U/L

***Within 48 hours of admission***

- ✓ Decrease in hematocrit >10 %
- ✓ Increase in blood urea nitrogen>2mg/dl
- ✓ Serum calcium<8mg/dl
- ✓ Fluid sequestration>4l
- ✓ Base deficit>5mmol/l

Further modification of this system in Glasgow by Imrie and his colleagues in 1978 led to the Glasgow system where only 9 factor need to be assessed[37]. A further refinement of this system by Blamey and Imrie in 1984 led to Modified Glasgow system where only 8 factor need to be assessed[38].

**MODIFIED GLASGOW CRITERIA[38]**

***within 48 hours of admission***

- ✓ Age >55 years
- ✓ White blood cell count>15000/$mm^3$
- ✓ Glucose> 180mg/dl
- ✓ Blood urea nitrogen> 45 mg/dl
- ✓ Lactate dehydrogenase > 600U/L
- ✓ Albumin< 3.2gm/ dl

- ✓ Arterial oxygen pressure < 60mm Hg

- ✓ Serum calcium <8 mg/dl

Despite refinement, the above scoring system were found to have low sensitivity and specificity[33,40]. Which has reduced the practical value of these scoring system for the early assessment of acute pancreatitis.

Another shortcoming with Ranson's is its inability to be used after 48 hours of admission and since acute pancreatitis is a dynamic process where repeated assessment of the patient is most desirable, Ranson's criteria are not helpful Other illness scores, which are used to qualify the severity of acute illness in intensive care units, have been used in acute pancreatitis with more efficacy than Ranson or Glasgow's criteria. They are:

- Simplified acute physiological score (SAPS)

- Acute physiological and chronic health evaluation score (APACHE II)

- Medical Research Council Sepsis score (MRCS)

## CT STAGING

The value of CT scan as an early predictive indicator of morbidity and mortality was first established by Sielgelmen et al in 1980 and Hill et al in 1982.

Balthazar in 1989 graded patient with acute pancreatitis into five categories based on CT scan finding[9]. He showed that patients without peripancreatic inflammation (grade A&B) have a mild uncomplicated course while those with one or more peripancreatic collection (grade D&E) often exhibit a protracted clinical illness with a higher frequency of complication and death.

**TABLE 3: CLASSIFICATION OF TYPE AND SEVERITY OF PANCREATIC INFLAMMATION BASED ON CT SCAN (BALTHAZAR 1989)[9]**

| | |
|---|---|
| **GRAD A** | Normal pancreas |
| **GRAD B E** | Restricted intraglandular changes with out evidence of peripancreatic disease, including non homogenous attenuation of the gland, contour irregularities, intrapancreatic foci of small fluid collections and |

| | |
|---|---|
| **GRAD E C** | Intrinsic pancreatic abnormalities associated with inflammation of peripancreatic fat giving it a hazy |
| **GRAD E D** | Single poorly defined extra pancreatic fluid collection or phlegmon |
| **GRAD E E** | Two or more ill- define extra pancreatic collection or the presence of gas in or adjacent to |

Another prospective study by the same author evaluated the prognostic significance of pancreatic necrosis as detected by CT scan.

The positive predictive value for development of pancreatic abscess was 84% in patient with necrosis and phlegmon ( grade D&E) , 77% in patient with necrosis alone and 46% in patient with phlegmon alone.

Other conclusion of this study were firstly, the degree of early necrosis does not seem to be of prognostic value and secondly late necrosis appearing during follow up signifies poor prognosis in patient with initially normal pancreatic enhancement.

This stressed the utility of follow up CT scan every 2 weeks in patient with peripancreatic phlegmons [29]

In another study comparing CT scan and APACHE II scoring, CT scan criteria best defined local anatomical abnormality and was found to be superior to APACHE II score as predictor of local complication, but CT criteria were inferior to APACHE II as indicators of systemic disease severity (as reflected in the need for ICU admission). The most effective initial triage would be APACHE II Calculation

Drawback of CT scan are the expenses, limited availability, limited specificity and inconvenience for several ill patient.

Rebeneck clinical prognostic staging system using clinical feature and comorbid illness is easy to use and requires no special or additional tests.

**TABLE 4: Clinical Severity of Acute Pancreatitis with Comorbidity**

| **Clinical Severity of Acute** | **Comorbidity[41]** | |
|---|---|---|
| | **Non** | **Sever** |
| No Ileus or Peritonitis | I | II |
| Ileus or Peritonitis | II | III |

Complication or death occurred in 7% of patient in stage I, 40% of patient in stage II, and 67% of patient in stage III.

Agarawal and Pitchumoni outline clinical assessment for severity stratification in which the main adverse criteria are 1-high quantity fluid requirement( 5 liters or more per day for the first two days) to maintain hemodynamic stability 2- early organ dysfunction, especially renal and pulmonary[42].

Recently Imrie and his colleagues have questioned the Atlanta classification system where in all patient with organ dysfunction are classified as having severe acute pancreatitis. They suggested that only patients with worsening organ dysfunction should be classified as having severe acute pancreatitis and they found that dynamic nature of early organ dysfunction is a predictor of acute pancreatitis.

***SINGLE PROGNOSTIC FACTOR***

Several clinical sign, biochemical marker and imaging procedure have emerged in an attempt at early identification of pancreatic necrosis, monitoring of its progression and assessment of the response to therapy.

In particular C- reactive protein, leucocyte elastase typsinogen activation peptide[44] and inter leukin-6 have shown promise as simple marker of disease severity.

Recently serum procalcitonin, a marker of systemic inflammation has been found to have sensitivity of 92% and specificity 84% at 24 hrs from onset of acute pancreatitis for predicting severity[48]. Another factor activation peptide of carboxypeptidase B in urine has been found to have sensitivity of 92% and specificity of 89% for predicting severity of acute pancreatitis[46]

## THE APACHE SYSTEM

Acute physiological and chronic health evaluation.

The first major attempt at a system to quantity severity of illness in ICU patient was the APACHE system, by Knaus et al in 1981[47].

### *APACHE I:*

In original form, APACHE contained 34 physiological measurement and included many continuous variables. A value of 0-4 was assigned to each variable, according to its degree of abnormality. Shortly after its introduction *APACHE I* system was disfavored, because of practical problem like collection of large number of variable. Also under the rule of APACHE system any unmeasured variable was assumed to be normal and weighted as zero. This gave rise

to question about the model general applicability. Another major criticism of original APACHE system was that the variables were chosen by a group of physician and hence there was potential of bias. These inaccuracies in the original APACHE system prevented its widespread use. However, it did serve as a prototype for the development of two subsequent systems.

***SAPS:***

The simplified acute physiological score was developed from APACHE I system and incorporated 13 variable that had the most discriminate power and were the most frequently measure variables to cover all major organ system. SAPS score is still used but has essentially been replaced by APACHE II in many centers.

**APACHE II**

Published in 1985 by the same author this is the second version of the APACHE system and it contain refinement based on experience with the original APACHE system. APACHEII has been extensively used and has received for more attention in the literature than any of the other methodologies for ICU Out comes prediction. It contain 12 continuous variables from the original APACHE system and also takes into account age of the patient, pre- morbid condition and Glasgow coma scale.

***DEVELOPMENT OF APACHE II :***

Using clinical judgment and documented physiological relationship to choose variables and assign weight remains the essence of APACHE II. The number of variables was reduced from 14 to 12. Infrequently measured variables such as serum osmolality, lactic acid level, and the skin testing for energy were deleted. Serum BUN was replaced by more specific serum creatinine and serum pH was retained in preference to bicarbonate. Many variables crucial in patient care, such as serum glucose, albumin, CVP and urinary output were found to have less explanatory power. Most of these variables were sensitive to variation in therapeutic decision than severity of disease.

Some of the threshold and weight for the physiology variables have been changed e.g. Glasgow coma score, serum creatinine. Also since alveolar- arterial O2 gradient(p [A-a] O2) is heavily dependent on inspired O2 (F1 O2) a direct weighting was given to all paO2 values when FiO2 is less than 0.5

To eliminate the problem of missing values and concerns about the assumption that an unmeasured variables was normal, measurement of all 12 variables was made mandatory for usage of APACHE II. The recorded value of the variables are based on the most deranged values during the past 24 hours.

Because age and severe chronic health problem reflect diminished physiological reserve, they have been directly incorporated into APACHE II. also, emergency surgery and non operative patient with severe chronic organ system dysfunction were given additional five points in comparisons to elective surgical patient who were given only two points because patient with severe chronic condition are not considered to be candidates for elective surgery.

The maximum possible APACHE II score is 71. in the experience of the author of APACHE II no patient had exceed 55.

The strengths of APACHE II system are

1. It has a well- define outcome (hospital death)

2. It was derived from a database (5815 patient from 13 hospital)

3. Source of bias present in its prototype was understood and corrected.

***SHORT COMINGS OF APACHE II SYSTEM***

Because of extensive usage, important sources of error and bias in the APACHE II system were revealed. First, APACHE II perform well over all in several ICU population but it is inaccurate when looking at specific disease categories because the data base from which it was derived, though large, did not contain many patient in major disease subsets such as cardiac surgery, oncology etc. second, APACHE II dose not accounts for prior treatment or clinical course before the patient enter ICU, this has been labeled as lead- time bias. Third, APACHE II require determination of a single admission diagnosis, a subjective process prone to bias. Finally despite reduction in number of variables, measurement error from bedside data collection is still on issue.

APACHE II has been recently refined into APACHE O, where O represents Obesity, and this is a better prognosis than APACHE II[48] . another modification of APACHE II is the APACHE III system, which is yet to be applied widely to acute pancreatitis clinical trial[49].

## ATLANTA CLASSIFICATION[7]

An international symposium was conducted in Sep 11 through 13, 1982, and a unanimous consensus on a series of definition and a clinically based classification system for acute pancreatitis was achieved by a diverse group of 40 international authorities from six medical disciplines and 15 countries. The proposed classification system is of value to practicing clinicians in the care of individual patient and to academicians seeking to compare inter institutional data.

The Atlanta symposium defined terms like acute pancreatitis (severe and mild), acute fluid collection, necrosis, pseudocyst and abscess. The symposium also discarded term like phelgmon, infected pseudocyst and hemorrhagic pancreatitis.

The present study makes use of these definition while predicting the patient out come.

## DEFINITION

### *ACUTE PANCREATITIS*

Acute pancreatitis is an inflammatory process of the pancreas, with variable involvement of other regional tissue or remote organ system.

***SEVERE ACUTE PANCREATITIS***

Severe acute pancreatitis is associated with organ failure and/ or local complication, such as necrosis, abscess or pseudocyst. This is characterized by three or more Ranson criteria or eight or more APACHE II points.

***MILD ACUTE PANCREATITIS***

Mild acute pancreatitis is associated with minimal organ dysfunction and an uneventful recovery, and it lacks the described feature of severe acute pancreatitis.

***ACUTE FLUID COLLECTIONS***

Acute fluid collection occur early in the course of acute pancreatitis, are located in or near the pancreas, and always lack a wall of granulation or fibrous tissue

***PANCREATIC NECROSIS***

Pancreatic necrosis is a diffuse or focal area (s) of nonviable pancreatic parenchyma, which is typically associated with peripancreatic fat necrosis.

***ACUTE PSEUDOCYST***

A pseudocyst is a collection of pancreatic juice enclosed by a wall of fibrous or granulation tissue, which arise as a consequence of acute pancreatitis, pancreatic trauma, or chronic pancreatitis.

***PANCREATIC ABSCESS***

A pancreratic abscess is a circumscribed intra-abdominal collection of pus, usually in proximal to the pancreas, containing little or no pancreatic necrosis, which arises as a consequence of acute pancreatitis or pancreatic trauma.

Phlegmon, infected pseudocyst, hemorrhagic pancreatitis and persistent acute pancreatitis are nonspecific terms in general usage, these should be discarded and specific terms as defined above should be used.

## TREATMENT OF ACUTE PANCREATITIS

### MEDICAL TREATMENT

Treatment of acute pancreatitis is primarily conservative. Basic medical therapy of acute pancreatitis includes withholding of oral feeding, nasogastric suction for paralytic ileus, and correction of fluid, acid base disorders, electrolytes and vigorous volume replacement. Non morphine based analgesia and sedative are often required for pain. In mild acute pancreatiits, antibiotics require only if disease is due to biliary stones, with attendant possibility of bile infection in more than 50% cases[50]. Early antibiotics prophylaxis is strongly advocated in acute necrotizing pancreatitis as the initial sterile necrosed tissue is vulnerable to infection because of its avascularity and the acute

catabolic infection. The incidence of infection is 20% in first week, 40% in second weeks and 60% in third weeks. Infection dramatically worsens the prognosis, with a mortality ranging from 20% to 50%.

Currently, majorities of death in necrotizing pancreatitis are associated with infection[51]. The organism responsible for infection traverse by translocation from intact gut and are the enteric flora mostly coliform, with 20%-30% anaerobes.

Imipenem is the antibiotic of choice, as ( i) it has the maximum blood- pancreas diffusion, and (ii) it has widest spectrum against organism of enteric flora, including anaerobes[52]. However , the cost factor may force the use of quinolones as the next best choice in combination with an anti- anaerobe.

Various well define studied have failed to demonstrate any impact on outcome of the disease with the use if somatostatin derivative of octreotide, however few smaller studies[53] have shown a trend towards decrease in complications.

Aggressive nutritional support should be initiated either as total parenteral nutrition or alternatively, early elemental jejunal feeds can be instituted to preserve or restore mucosal barrier and gain immune therapeutic benefit.

Therapeutic cytokine manipulation using anti TNF antiserum, CNI-1493 and AF antagonist Lexipafant[54] are under clinical trials and have been shown to decrease the severity of acute pancreatitis and reduced the incidence of multiorgan system failure. A therapeutic window, 12- 36 hours after onset if pain, has bees shown to exist allowing for the antagonism of cytokines during clinical pancreatitis.

It is a known fact that the only definitive treatment of a patient with acute pancreatitis in the initial 72 hrs is massive fluid transfusion to maintain hemodynamic stability[6]. All other modes of treatment offered to the patient from nasogastric aspiration to surgery have been debated and their effectiveness has been questioned[55,56,57].

Massive 3rd space fluid loss may be primarily responsible for tissue anoxia, its may aggravate already existing cellular anoxia further.

## PROPOSED NONOPERATIVE THERAPIES FOR ACUTE PANCREATITIS

### *SUPPORTIVE MEASURES*

- ✓ Intravenous fluid therapy
- ✓ Electrolyte replacement
- ✓ Analgesia
- ✓ Nutritional support
- ✓ Antibiotics
- ✓ Respiratory support

## PANCREATIC EXOCRINE SECRETION SUPPRESSION

- ✓ Nasogastric suction
- ✓ Hitamine ((H2) receptors antagonists
- ✓ Antacids
- ✓ Anticholinergies
- ✓ Glucagon calcitonin
- ✓ Somatostatin
- ✓ Peptide YY
- ✓ Cholecystokine receprors antagonists

**PANCREATIC ENZYME INHIBITION**

Protease inhibitors:

- ✓ Aprotitin
- ✓ Gabexate
- ✓ Camostae

Fresh frozen plasma

Antifibrinolytic

Chloroquine

PhaspholipaseA

inhibitors

**PANCREATIC PROTECTION FROM OXYGEN DERIVED FREE RADICALS**

- ✓ Free radicals scavengers
- ✓ Xanthine oxidase inhibitors
- ✓ Isovolemic hemodilution

**EXAMINATION OF TOXIN INTRAPERITONEAL COMPOUNDS**

Peritoneal dialysis

**PLATELET- ACTIVATION FACTOR ANTAGONISTS**

Lexipafant

***SURGICAL TREATMENT***

Indication for operative management in patient with severe acute pancreatitis[58]

- Persisting acute abdomen
- Infected necrosis
- Sterile necrosis causing MODS syndrome ( not responding to maximum ICU measures for more than 72 hours)
- Correction of associated biliary tract disease
- Persisting and increasing local complications
    - ✓ Pancreatic abscess
    - ✓ Pseudocyst
    - ✓ Gastrointestinal fistula

**TABLE 4: ATLANTA CLASSIFICATION AND ITS MANAGEMENT**

| ATLANTA | TREATMENT PRINCIPLES |
|---|---|
| Interstitial- edematous pancreatitis (mild AP) | Non surgical |
| Necrotizing pancreatitis (severe AP)<br>• STERILE necrosis<br>• Infected necrosis | Non surgical ,surgical if non Surgical debridement+continuous lavage and open packing |
| Pancreatic abscess | Interventional drainage, in case of persisting sepsis- surgical drainage |
| Acute pseudocyst | Interventional drainage, surgical drainage is second choice. |

**Fig 17:- Intraoperative Pancreatic Pseudocyst**

**Fig 18:- CECT – Pancreatic Psudocyst**

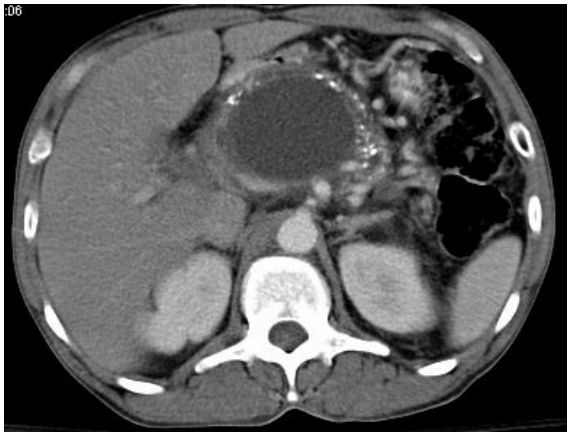


**Fig 19:- USG – Pancreatic Psudocyst**

**Fig 20:- CECT – Pancreatic necrosis**

**Fig 21(a):- Intra Operative- Pancreatic Necrosis**

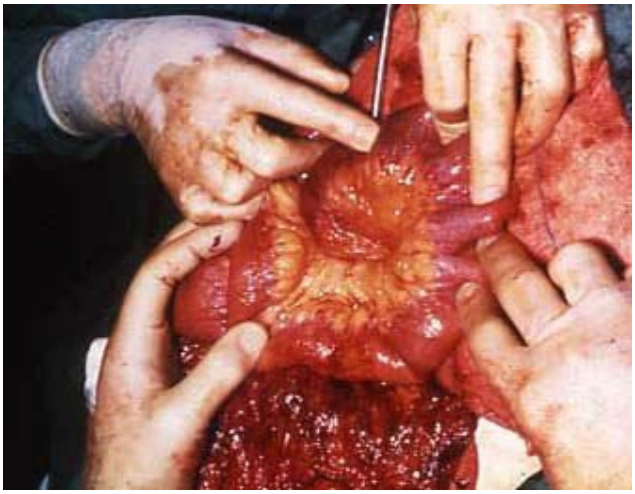
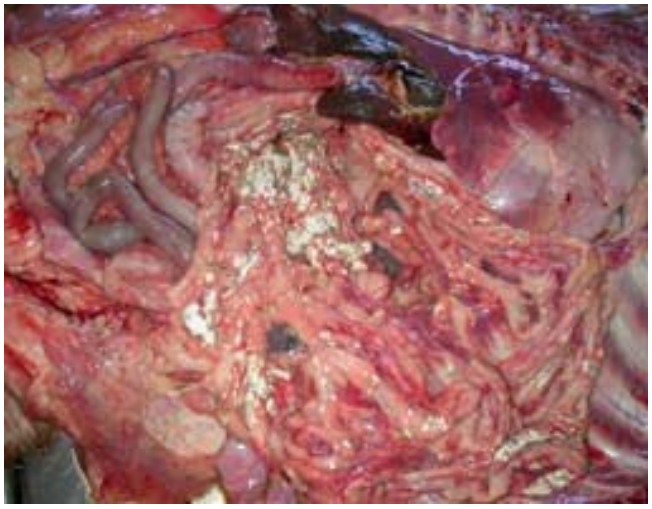


**Fig 21(b):- Specimen showing Pancreatic Necrosis**



## UNCERTAINTY OF CLINICAL DIAGNOSIS

Because no single test or combination of studies can enable one to diagnosed acute pancreatitis with 100% accuracy, it may occasionally be difficult to exclude other diagnoses that mimic acute pancreatitis and require operative intervention example of such condition include perforation viscus and acute mesenteric ischemia. In these situation when the clinical diagnosis not confirm, exploratory laparotomy or diagnostic laparoscopic may be indicated to exclude a surgically correctable disease with a potentially fatal outcome in the unoperated state. If uncomplicated acute pancreatitis is present, no manipulation is indicated, and the operation is terminated. In patient with cholelithiasis and presumed gallstone- associated pancreatitis, definitive biliary surgery, is favored if the clinical circumstances permit. In patient with severe necrotizing pancreatitis without frank infection, formal pancreatic resection is not favored. Instead, caution debridement of necrotic tissue is performed, and wide retroperitoneal drainage is considered.

## TREATMENT OF SECONDARY PANCREATIC INFECTION

Pancreatic abscess, infected pancreatic pseudocyst, and infected pancreatic necrosis are three life threatening complication of acute pancreatitis that occur in up to 5% of all patient. By definition, pancreatic abscess and infected pancreatic pseudocyst are associated with little or no necrosis. The combination of abdominal USG/CT scan and guided percutaneous needle aspiration (and bacteriology) of fluid (from three different areas to avoid sampling error) has been demonstrated to be highly reliable in differentiating a pancreatic infectious process from a sterile necrotic collection[59]. Treatment of secondary pancreatic infection combines antibiotic therapy with prompt drainage. Operative debridement is usually necessary to remove the thick, debris- filled. Paste like collection of infected necrotic materials in patient with infected pancreatic necrosis. The two currently acceptedoperative management of infected pancreatic necrosis and some pancreatic abscess are

1. Laparotomy with debridement and wide sump drainage, and
2. Laparotomy with debridement, open packing, and scheduled repacking[5,60].

A recent technical is "closed laparostoma" this is a semi-open method of temporary closure of abdomen with zipper, Gortex,

Marlex or Nylon[61] in order to avoid very frequent scheduled reoperation and to reduce loss of fluid, electrolyte and protein associated with open packing. In both these method relaparotomy and redebridement are carried out until the progressive necrotic process is controlled and no further infection/ necrosis is evident.

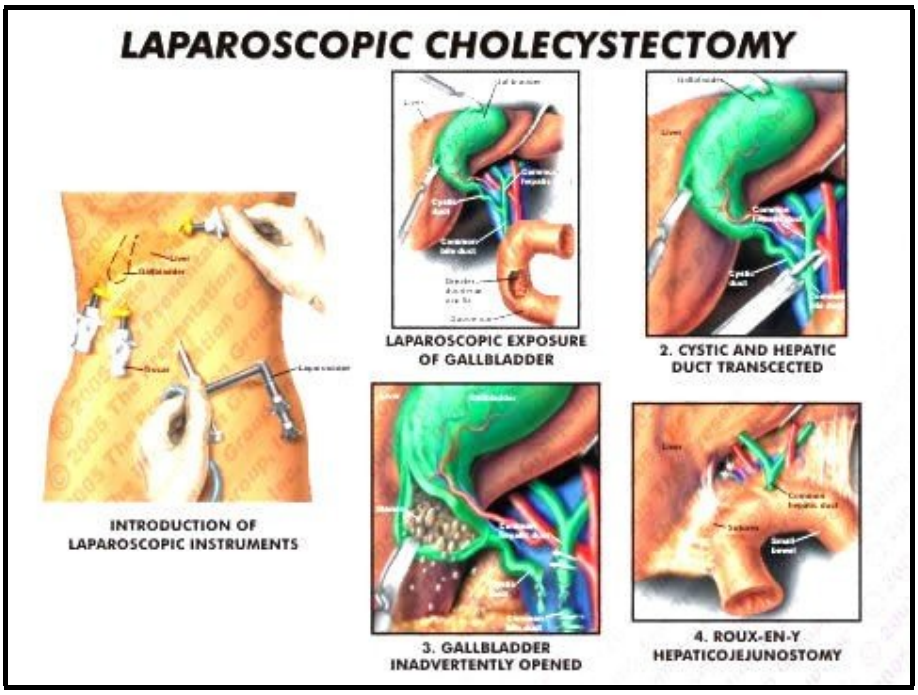
Recently peripancreatic necrosis collection has been treated by retroperitoneal route using endoscopic techniques[62]. With encouraging preliminary result. However this approach is not exclusive and may be combined with a secondary laparotomy when needed.

***CORRECTION OF ASSOCIATED BILIARY TRACT DISEASE***

Formerly, definitive biliary tract operation for gallstone - associated pancreatitis were often deferred up to 8 weeks after the acute episode of pancreatitis. This approach gradually lost favour, primarily because of the advent of laparoscopioc cholecystectomy and because the natural history of gall- stone associated pancreatitis without surgical intervention is that of frequent recurrence. Because most episode of gallstone- associated pancreatitis followed a mild, short lived clinical course, and because definitive biliary tract operation at the time of the index admission have proved safe and cost effective, early biliary tract procedure are now favored in most patient. Most surgeon proceed with laparoscopic cholecystectomy, after clinical resolution

of pancreatitis and normalization of the liver function test.

**Fig 22:- Laparoscopic Cholecystectomy**




Three exception to this treatment algorithm for gallstone pancreatitis are

1. Gallstone pancreatitis and either jaundice or cholangitis
2. Severe acute pancreatitis
3. Patient who refuse surgery or are at extraordinary high risk for elective cholecyctectomy.

Patients with severe acute pancreatitis, especially those with obstructive jaundice or cholangitis, the currently recommended treatment includes urgent ERCP and sphincterotomy. Studied have

shown that patient with predicted severe pancreatitis are more likely to benefit from endoscopic intervention that those with predicated mild pancreatitis[30,31,32,33,34,35].

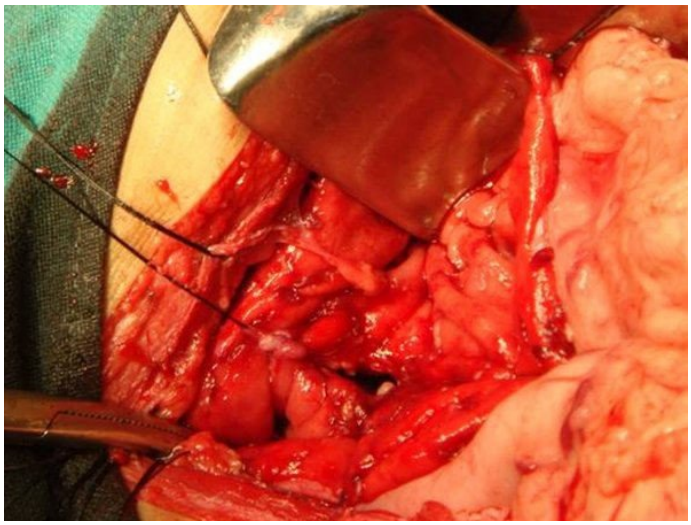
***TREATMENT OF COMPLICATIONS***

Pancreatic abscess is encountered less frequent than infected pancreatic necrosis. Currently, the treatment of choice is percutaneous USG/CT guided aspiration and drainage by one or more pigtail or sump drains[63]. But in 30% to 50%, it may fail or not advisable because of considerable necrosis, multilocularity or severely ill patient.

Acute pseudocyst is seen in 5-10% of patient with acute pancreatis . about 40% of these, especially those without ductal communication and those with are less than 5-6 cm in diameter resolve spontaneous. A pseudocyst lead to pressure effect before at least 6 weeks is best treated by an initial percutaneous USG/CT guided therapeutic aspiration. The aspiration is followed by ERCP, and if pancreatic ductal leak is demonstrated, the duct is stented. If these procedure fails, the cyst is drain to exterior by conventional or laparoscopic surgery. Observation beyond 6 weeks permits maturation of the cyst wall. At this point of time, several treatment option are available. A cyst less than 5 cm in diameter is just observed. Cyst more than 5 cm in diameter are drained into jejunum, stomach, or

duodenum, depending on the local anatomy: for this internal drainage, laparoscopic surgery is slowly superceding conventional surgery (and is practiced successfully by increasing number of Indian surgeon, notable among these being Udwadia, Chowbey, Palanivellu and Thanakumar). A current non-surgical option is endoscopic transgastric or transduodenal stenting of the cyst[51].

**Fig 23:- Intraoperative view of Cystogastrostomy**



Gastrointestinal fistula is a rare complication and are due to autodigestive necrotic process. Upper left colon is the most frequent site. The left colonic fistula is best managed by diversion loop ileostomy( transverse mesocolon is usually severely edematous and foreshortened)

At times, fistula is multiple. Development of gastrointestinal fistula adversely affects prognosis [64,65].

Pancreatic ascites is managed by repeated ascetic tap, TPN, somatostatin or octreotide to reduced pancreatic secretion are tried as conservative therapy for 3 weeks.

If there is no improvement beyond 3 weeks, ERCP stenting of pancreatic duct is done initially. Then resection or drainage surgery done. Pancreatic pleural effusion disappear once abdominal cause or ascites is corrected.

## METHODS AND MATERIALS

The study group has evaluated 100 consecutive patient with clinical, biochemical and radiological diagnosis of acute pancreatitis admitted to hospitals attached to Madras Medical College between March 2014 to August 2014 . It's a prospective Hospital base study.

**INCLUSION CRITERIA:**

- Patients who provisionally diagnosed as acute pancreatitis

**EXCLUSION CRITERIA:**

- Patient who provisionally diagnosed as chronic pancreatitis

All the patients were evaluated thoroughly at the time of admission and frequently at the in those showed deterioration their clinical status to find out associated local/ systemic complication

Blood sample for laboratory studied on admission was drawn in the ICU / general ward prior to intravenous infusion.

Initial conservative management consists of nasogastric suction, intravenous administration of fluid, antibiotic and supportive care in all patient.

An indwelling urinary catheter was placed in most patient to allow close monitoring of urine output, and a CVP catheter was frequently introduced.ancreatitis

- Development of systemic complication

1. Shock(systolic BP <90 mmHg)
2. Pulmonary insufficiency PO2< 60mmHg or less require ventilation or O2 therapy.
3. Renal failure(output<400ml/24 Hours), serum creatinine>2mg%
4. Severe metabolic disturbance(serum calcium7.5mg/dl or less)

- Development of local complication
  - Pancreatic pseudocyst
  - Pancreatic necrosis
  - Pancreatic abscess
  - Pancreatic ascites, pleural effusion, fistula

***STATISTICS***

Data was processed using excel software programme observation are represented as Bar diagram and Pie chart.

## RESULTS

A total of 100 consecutive patient who provisionally diagnosed as acute pancreatitis were entered in the study group. All had an admission of acute pancreatitis and satisfied the inclusion criteria.

***TYPE OF COMPLICATIONS***

Out of 100 patients 31(31%) developed only one local complications, 7(7%) developed isolated systemic complication,10 (10%)developed more than one local complication and 12(12%) developed both systemic and local complication

***SEX DISTRIBUTION***

Out of 100 patients 93(93%) were male and 7 (7%) were female. Sex ratio is 13.2: 1.

Out of 93 males 31 developed single local complications and 11 had both systemic and local complication and 6 developed only systemic

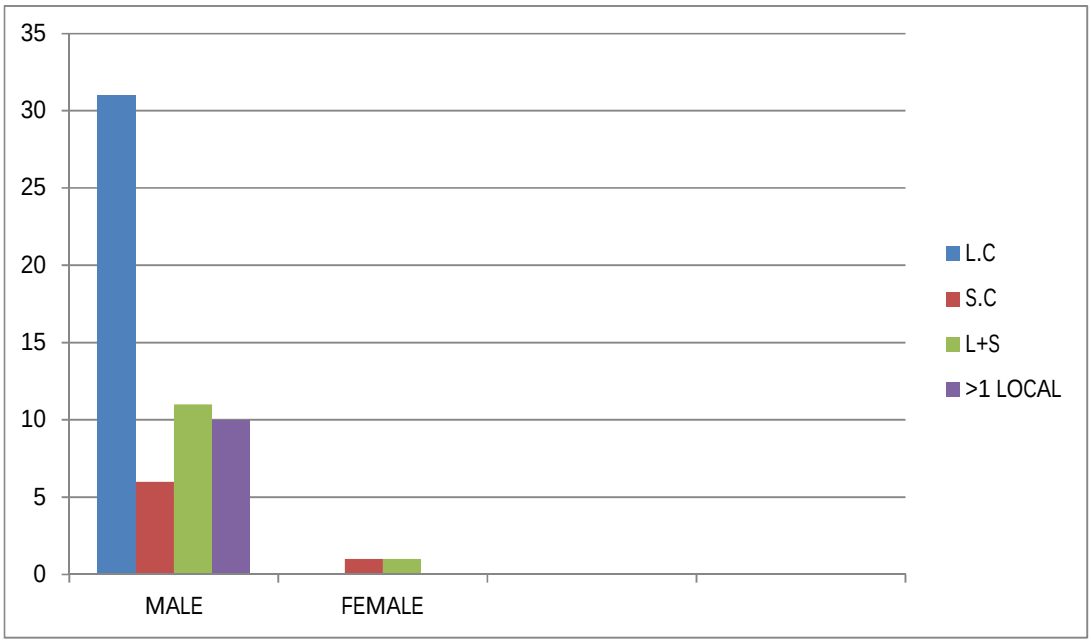
complications.

Out of 7 female 1 developed both systemic and local complication and 1 developed systemic complications

**Table 6 : Sex Distribution**

| SEX | LOCAL COMP. | SYSTEMIC COMP. | BOTH | >1 LOCAL COMP. | TOTAL | PERCENT AGE |
|---|---|---|---|---|---|---|
| MALE | 31 | 6 | 11 | 10 | 58 | 58% |
| FEMALE | 0 | 1 | 1 | 0 | 2 | 2% |
| | | | | | | |
| TOTAL | 31 | 7 | 12 | 10 | 60 | 60% |

**Fig 24:- Sex Distribution**



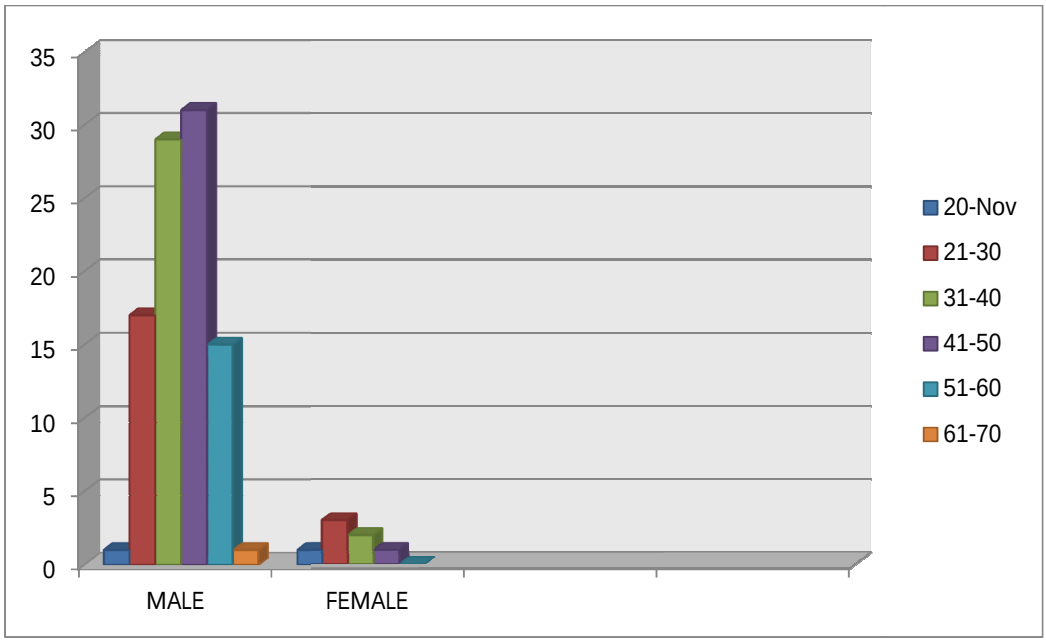
## Age distribution

The peak incidence was in the 5th decade in male (33%) and 2nd decade in female. (42%). The mean age group in our study is 40.43 years.

**Table 7 : Age distribution**

| Age group in years | No of male | No of female |
|---|---|---|
| 11-20 | 1 | 1 |
| 21-30 | 17 | 3 |
| 31-40 | 29 | 2 |
| 41-50 | 31 | 1 |
| 51-60 | 15 | 0 |
| 61-70 | 1 | 0 |

**Fig 25:- Age distribution**



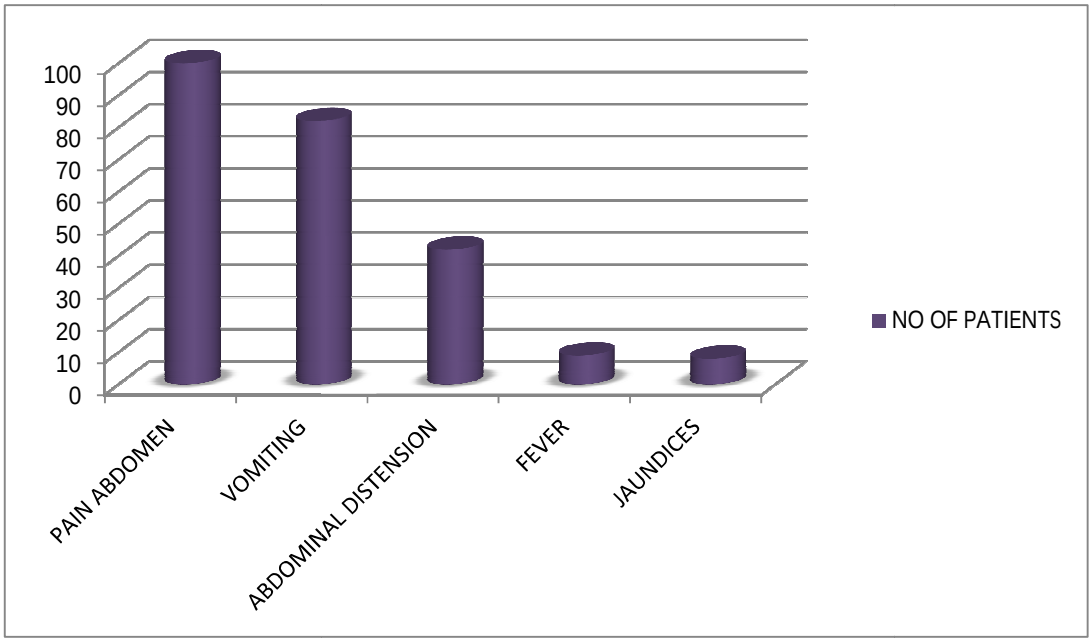
### *Clinical feature*

Pain abdomen was the presenting complaint in almost all of the 100 patient Other clinical feature includes vomiting in 82, distention in 42, fever in 18, jaundice in 8 and.Patients Cullen's sign and grey turner sign was seen in 6 cases associated with severe necrotizing pancreatitis.

**Table 8 : Clinical Features**

| CLINICAL FEATURE | NO OF PATIENT | PERCENTAGE% |
|---|---|---|
| ABDOMINAL PAIN | 100 | 100% |
| VOMITING | 82 | 82% |
| ABDOMINAL DISTENSION | 42 | 42% |
| FEVER | 9 | 9% |
| JAUNDICE | 8 | 8% |

**Fig 26:- Clinical Features**



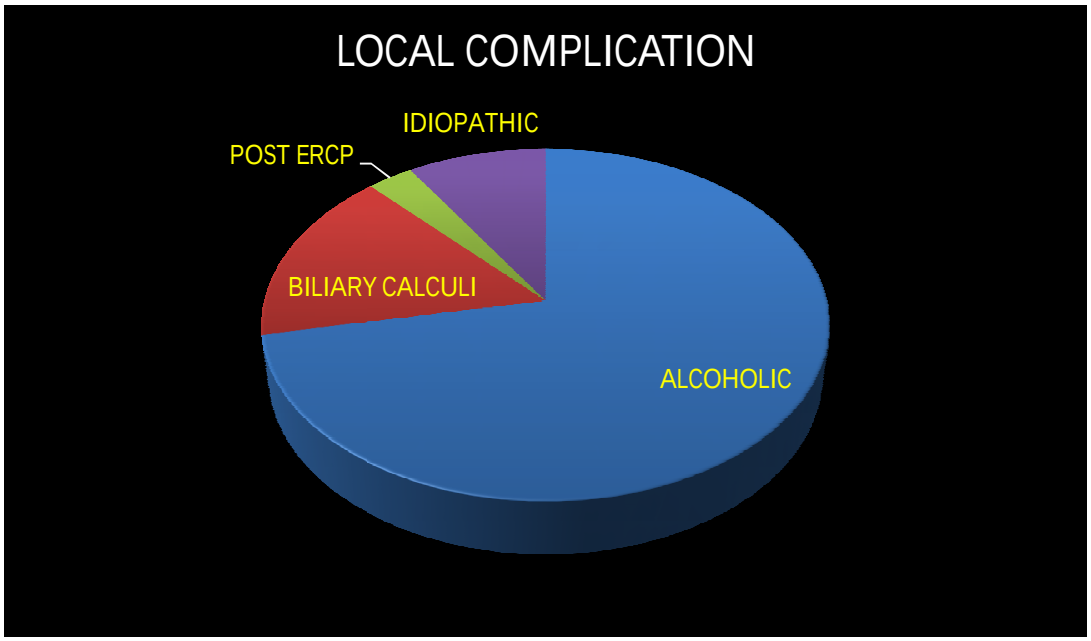
## ETIOLOGY

The history of alcohol consumption and like hood of it being the etiological factor was in7 2 patients. While gallstone were implicated in 16 patients and 3 patients developed pancreatitis after ERCP and 9 had no obvious cause

**Table 9 : ETIOLOGY**

| Etiology | No of patient (n=100) | Percentage % |
|---|---|---|
| Alcoholic | 72 | 72% |
| Biliary calculi | 16 | 16% |
| Post ERCP | 3 | 3% |
| Idiopathic | 9 | 9% |
| Total | 100 | 100% |

**Fig 27:- ETIOLOGY**



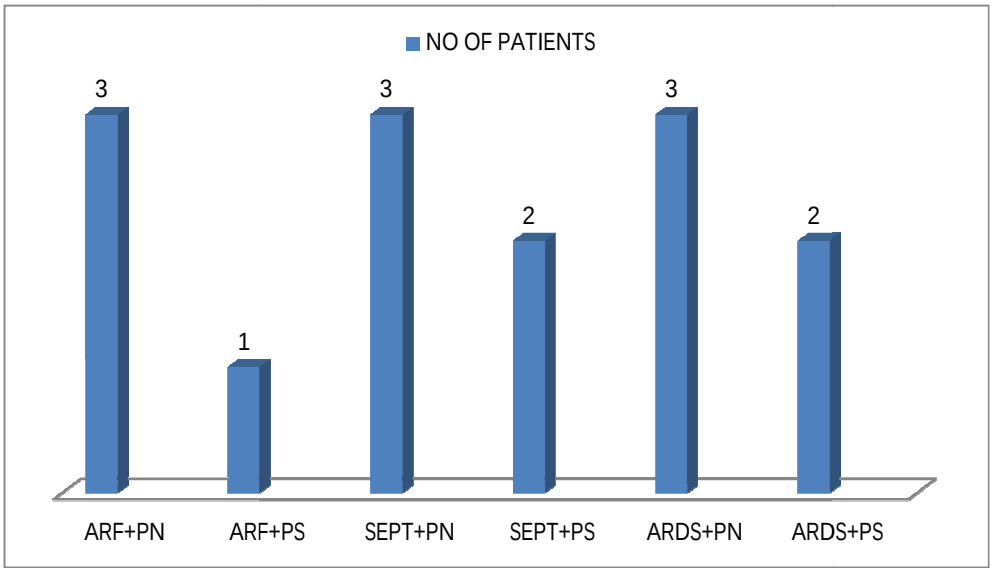
## *COMPLICATIONS*

All the 50 patients evaluated Clinically, Biochemically and Radio logically and found to have only one local complications in 31 patients and systemic complications alone in 7patients, both local and systemic complication in12 patients ,and more than one local complication in 10 patients.

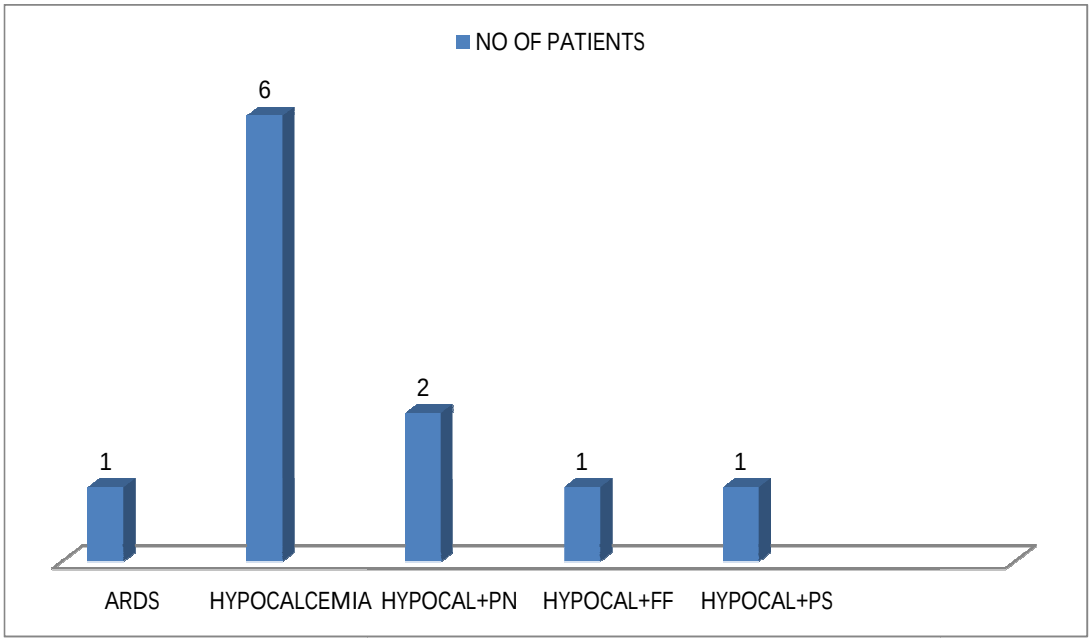
**Table 10 : SYSTEMIC COMPLICATIONS**

| Systemic complications | No of patients |
|---|---|
| ARF+PANCREATIC NECROSIS | 3 |
| ARF+PSEUDOCYST | 1 |
| SEPTICEMIA+PANCREATIC NECROSIS | 3 |
| SEPTICEMIA+PSEUDOCYST | 2 |
| ARDS+PANCREATIC NECROSIS | 3 |
| ARDS+PSEUDOCYST | 2 |
| HYPOCALCEMIA+PANCREATIC NECROSIS | 2 |
| HYPOCALCEMIA+PSEUDOCYST | 1 |
| HYPOCALCEMIA+ASCITIS | 1 |
| HYPOCALCEMIA ALONE | 6 |
| ARDS ALONE | 1 |

**Fig 28:- SYSTEMIC COMPLICATIONS**



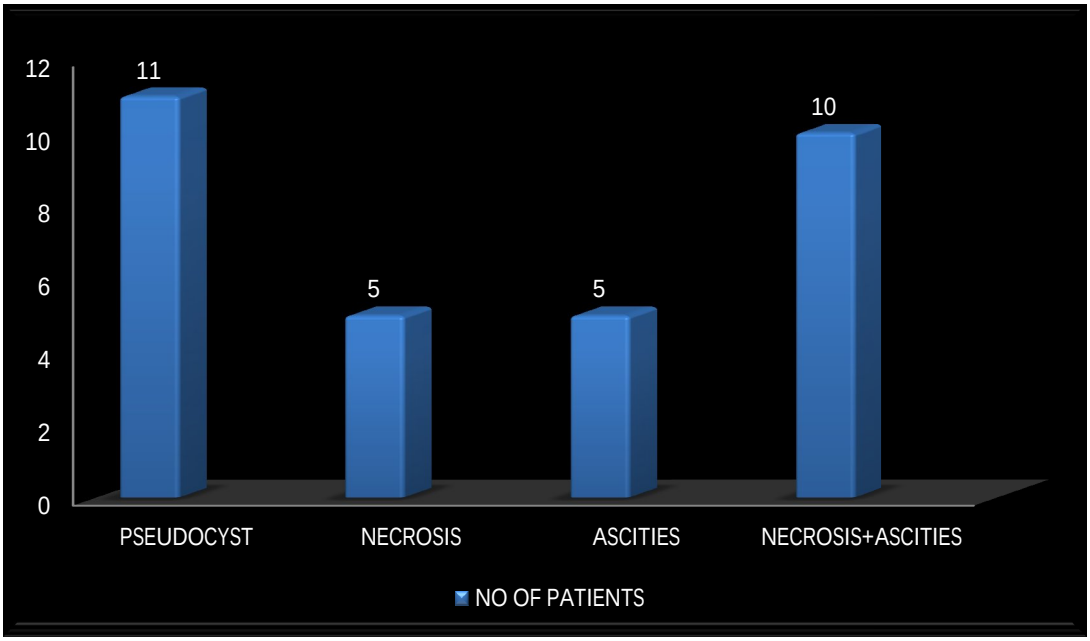
## SYSTEMIC COMPLICATION CONT…



## TABLE 11: LOCAL COMPLICATIONS

| LOCAL COMPLICATIONS | N-31 |
|---|---|
| NECROSIS | 5 |
| PSUDOCYST | 11 |
| ASCITES | 5 |
| NECROSIS+ASCITIES | 10 |
| TOTAL | 31 |

## Fig 29:- LOCAL COMPLICATIONS



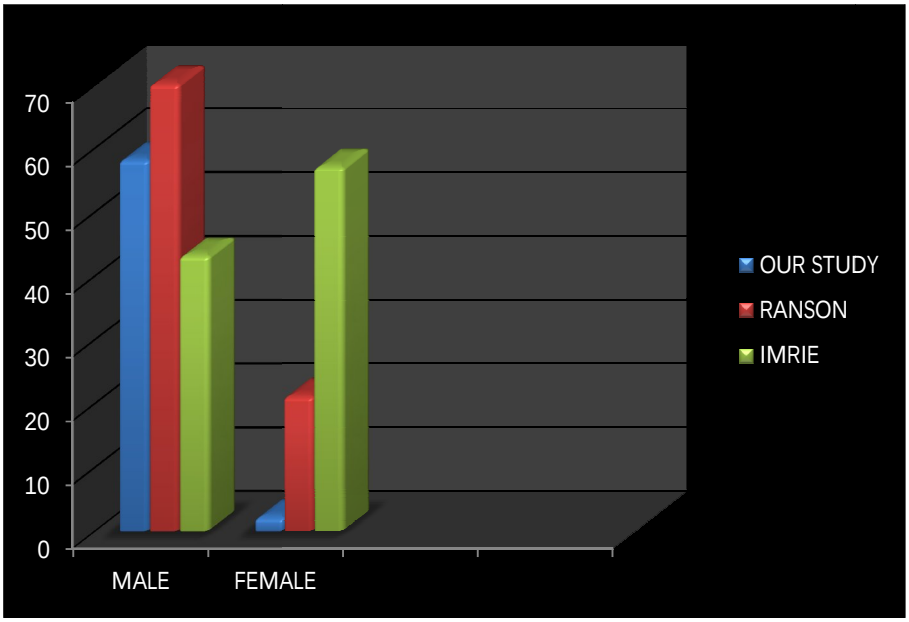
# DISCUSSION

The early identification of potentially severe acute pancreatitis enables the selection of patients who may require more intensive and invasive method of management than are appropriate in mild pancreatitis. Most of the patients with associated with systemic complication were managed in general ward expect few patients who are associated with septicemia and ARDS.

In this study laboratory test done are simple, routine and readily available. These investigations were used to identifying systemic complication. Local complication was diagnosed by USG/ CT scan.

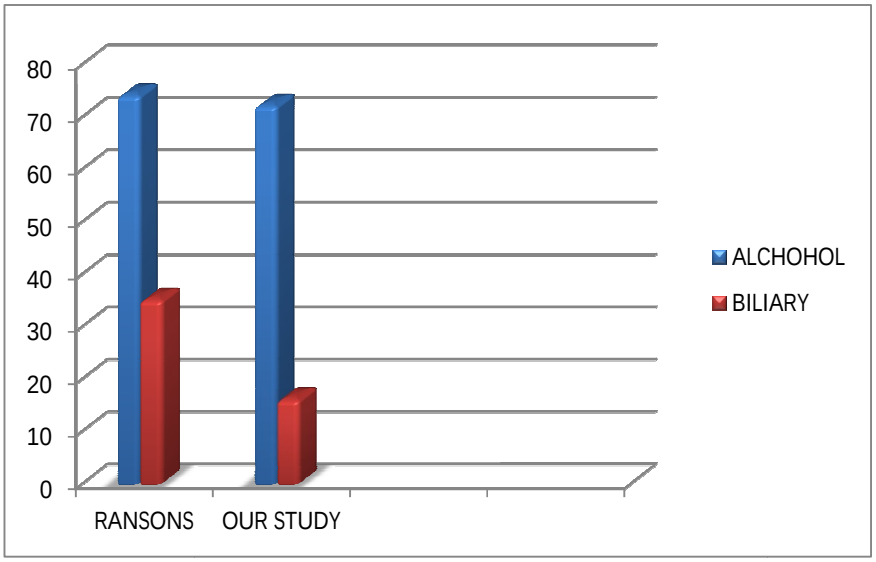
In this study complication was 21.5 times more common in male than female (M:F:21.5:1 In Ranson study male sex incidence is higher (M:F=3.7:1) where in Imries study female sex incidence is higher (M:F=1:1.3) the male sex incidence in study is higher because of higher incidence of alcoholic pancreatitis. In India consumption of alcohol in female is very low compare to western countries.

**Fig 37:- SEX DISTRIBUTION**




The mean age group in study 40.3 years. This is near the common mean age in the 4$^{th}$ decade in other study also complication are more common in Female in this study Alcohol is found to be more common etiological factor accounting for 72% of total cases followed by gall stone 16%. This is consistent with Ranson study where alcohol factor was found to be higher 80% as compare to Imrie study where gall stone was higher.

**Fig 38:- ETIOLOGY**




Regarding clinical feature all the patients were presents with acute abdominal pain followed by vomiting and distention. In our study patients present with acute abdominal associated with raised serum lipase level.

Out of 100 patients, 48 patients were raised serum lipase level >5 times where rest patients had 2-3 times higher. Serum amylase level raised in 36 patients, 28 patients had normal serum amylase level.

TC was elevated >20,000 in 17 patients those were associated with pancreatic necrosis, abscess and septicemia and ARDS. Blood urea and creatinine raised in almost all patients

In our study patient who were diagnosed to have acute pancreatitis (raised serum lipase) was evaluated with serum lipase,

basic blood investigation, chest x ray to find out associated complication. Erect abdominal x ray was not much helpful in diagnosis, but it had a advantage to rule out acute abdominal condition like perforation and intestinal obstruction.

In our study USG abdomen done in all patients but at the time of admission inconclusive in most of patients because pancreas could not be visualized due to full bowel loops.

USG was helpful to identify local complications such as pseudo cyst and abscess. But it had the added advantage of being noninvasive and could be repeated at frequent interval when require and also as bed side procedure. It was inaccurate in detecting pancreatic necrosis but could localized pancreatic edema, per pancreatic fluid collection, gall stone, biliary sludge, ascites and pseudo cyst.

CT scan in this study was a very sensitive non invasive tool in diagnosis of pancreatitis and associated complication. Contrast enhanced could differentiate necrosis.

Regarding management all patients, diagnosed to have acute pancreatitis evaluated clinically, laboratory and radio logically. According to complication and severity patient were managed in general ward and ICU.

81% patients with systemic complication were managed in general ward and 19% in ICU those associated with septicemia and

ARDS.

Conservative management includes NPO, iv fluid, antibiotics. Analgesic, TPN and electrolyte imbalance. The average fluid requirement was 5L/day.regarding antibiotics 3rd generation cephalosporins (cefotaxime) were given to all patients with systemic complication admitted in general ward and Piperacillin + Tazobactum those associated with septicemia and ARDS. Use of cefotaxime decreases the chance of infection in local complication.

# CONCLUSION

Acute pancreatitis is one of the common differential diagnoses of acute abdomen especially in alcoholic and biliary calculi. Acute pancreatitis patients present with acute onset of abdominal pain radiates to back, associated with raised serum lipase.

Acute pancreatitis patients should be evaluated thoroughly clinically, blood investigation and radiological as this condition is associated with many complication both systemic and local. Early diagnosis is essential to manage the complication and decrease mortality.

Most systemic complications are associated with hypovolemia and systemic infection where as local complications are associated with pseudocyst/necrosis. Serum lipase is elevated in all patients 2-3 times and >5 times in pancreatic necrosis.

Investigations of complicated pancreatitis required hematological and biochemical investigation to identify systemic complications where USG/CT needs to identify local complications

Management of complicated pancreatitis consists of supportive care and conservative management for systemic complication. Local complications are managed by conservative as well as operative procedure. Most pseudocyst < 6 cm size is managed by USG abdomen follow up. Pancreatic necrosis is managed by nacrosectomy+ close drain. Abscesses were managed by USG/CT guided aspiration.

## SUMMARY

- The study includes a total of 100 patients of complicated pancreatitis. 93 male and 7 female.
- The peak incidence in male is $5^{th}$ decade in life and in female $2^{rd}$ decade in life.
- Alcohol accounts 72% total cases where as gall stone contributes 16%,idiopathic in 9% and post ERCP 3%
- Serum lipase raise in 2-3 fold in all patients expect in pancreatic necrosis where serum lipase raise in 5 fold.
- All the patients were investigated to find out complication( systemic/ local)
- Systemic complications were diagnosed by routine blood investigation, RFT, LFT, serum calcium and chest X ray.
- Local complications were diagnosed by USG abdomen and CT scan.
- 31 patients found to have only one local complications, 10 patients had more than one local complications,12 patients had both local and systemic complications and 7 patients had only systemic complication out of 7 patient with systemic complications only one patient died of ARDS.
- Most systemic complication were managed in general wards except few patients in ICU those were associated with septicemia and ARDS.

- Systemic complication were managed with supportive and conservative
- Local complications were managed with conservative and operative procedure.

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## PROFORMA

**PATIENT DETAILS:**

**ON ADMISSION:**

**Name:** **Age:** **Sex:** **IP No. :** **Date:**

**HISTORY:**

1. **Duration of abdominal pain –**

2. **H/o pain radiating to back- yes/no**

3. **H/o vomiting- yes / no**

4. **H/o Anorexia- yes / no**

5. **H/o fever - yes / no**

6. **H/o pale clay color stool - yes/ no**

7. **H/o abdominal distension – yes/no**

8. **H/o alcohol intake - yes /**

   **If yes-amount and duration**

9. **H/o drug intake - yes / no**

10. **If yes – name of drug/duration/dose**

11. **H/o any ERCP procedure- yes / no**

12. **H/o similar illness in past- yes / no**

**CLINICAL EXAMINATION:**

**VITAL SIGNS**

**Pulse:** **BP:** **Temp:**

**SYSTAMIC EXAMINATION**

**CVS :**

**RS :**

**ABDOMEN ;**

**INSPECTION ;**

**PALPATION ;**

**AUSCULTATION ;**

**PR;**

**INVESTIGATIONS :**

**Hemogram:**

**Renal Function Test:**

**Liver Function Test;**

**Sr.amylase & lipase:**

**Fasting lipid profile;**

**Sr.calcium;**

**Ultrasound:**

**CXR:**

**CECT abdomen;**

**Other;**

**PROVISIONAL DIAGNOSIS-**

**MANAGEMENT**

- **NPO**
- **IVF**
- **ANALGESIC**
- **Inj.Pantoprazole**
- **Inj.octreotide**
- **Antibiotic**
- **Vital monitoring**

**CONDITION ON DISCHARGE**

**FOLLOW UP;**

## KEY TO MASTER CHART

PE - Pancreatic Enlargement

PN -Pancreatic Necrosis

PS -Pseudocyst

FF - Pancreatic Ascites

BC - Biliary Calculi

PN - Pancreatic Necrosis

CECT - Contrast Enhancing Computed

Tomography

USG - Ultrasonography

N - NORMAL FATING LIPID PROFILE

E - ELEVATED FASTING LIPID PROFILE

N - No follow up

NC - No complaint

RAB - Recurrent Abdominal Pain

HC - Hypocalcemia

NC – No complications

NFU – No follow up

ID - Idiopathic

BI - Biliary

| S.NO | NAME | AGE | SEX | COMPLAINT | DURATION | ALCOHOL INTAKE | JAUNDICES | DRUG INTAKE | TRAUMA | PROCEDURE | FEVER | FAMILY H/O | SR.AMYLASE | SR.LIPASE | USG ABDOMEN | FAST.LIPID PROFILE | SR.CALCIUM | CECT ABDOMEN | ETIOLOGY | LOCAL COMPLICATION | SYSTEMIC COMPLICATION | DURATION OF STAY | FOLLOW UP |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | SIVAGNANAM | 32 | M | AP+AD | 2 | YES | NO | NO | NO | NO | NO | NO | 1876 | 2134 | PE+FF | E | 9.5 | PE+FF | AL | PN+FF | ARDS | 15 | NFU |
| 2 | BABU | 49 | M | AP+VO | 3 | YES | NO | NO | NO | NO | NO | NO | 384 | 455 | PE | E | 9.2 | PE | AL | | | 10 | NFU |
| 3 | MALAR | 26 | F | AP+VO+AD | 5 | NO | YES | NO | NO | NO | YES | NO | 689 | 986 | BC+FF | N | 8 | BC+FF+PE | BI | FF | HC | 7 | RAB |
| 4 | RAMESH | 31 | M | AP+VO+AD | 6 | YES | NO | NO | NO | NO | NO | NO | 876 | 987 | PE+PS | N | 8.7 | PE+PS | AL | PS | | 14 | NC |
| 5 | RAJAMANIKKAM | 52 | M | AP+VO | 3 | YES | NO | NO | NO | NO | NO | NO | 789 | 765 | PE | E | 9.2 | PE | AL | | | 4 | NC |
| 6 | IYYANNAR | 39 | M | AP+VO+AD | 4 | YES | NO | NO | NO | NO | NO | NO | 1433 | 1456 | PE+PS | E | 9.1 | PE+PS | AL | PS | | 8 | RAB |
| 7 | BABU | 42 | M | AP+VO | 4 | NO | YES | NO | NO | NO | NO | NO | 102 | 658 | BC | E | 9.5 | BC+PE | BI | | | 6 | NC |
| 8 | RADHAKRISHNAN | 33 | M | AP+VO | 3 | YES | NO | NO | NO | NO | YES | NO | 988 | 1409 | PE | E | 8.9 | PE | AL | | | 7 | NFU |
| 9 | VENKAT | 30 | M | AP+VO+AD | 5 | YES | NO | NO | NO | NO | NO | NO | 1690 | 2009 | PE+FF | E | 9 | PE+FF+PN | AL | PN+FF | | 13 | RAB |
| 10 | MURUGAN | 50 | M | AP+VO | 2 | YES | NO | NO | NO | NO | NO | NO | 1807 | 2097 | PE | E | 9.6 | PE | AL | | | 8 | NC |
| 11 | SEKAR | 45 | M | AP+VO | 3 | NO | YES | NO | NO | NO | NO | NO | 679 | 896 | BC | N | 9.1 | BC+PE | BI | | | 7 | RAB |
| 12 | ANTHONY | 32 | M | AP | 3 | YES | NO | NO | NO | NO | NO | NO | 890 | 1008 | PE | N | 8.9 | PE | AL | | | 5 | NC |
| 13 | MUTHUSAMY | 50 | M | AP+VO+AD | 4 | YES | NO | NO | NO | NO | NO | NO | 1378 | 1678 | PE | E | 7.9 | PE+PS | AL | PS | HC | 10 | RAB |
| 14 | MANIKANNDAN | 30 | M | AP+VO+AD | 5 | YES | NO | NO | NO | NO | YES | NO | 1876 | 1987 | PE+PN | N | 8.9 | PE+PN | AL | PN | | 15 | RAB |
| 15 | SHANMUGAM | 26 | M | AP | 3 | NO | NO | NO | NO | NO | NO | NO | 567 | 879 | N STUDY | N | 9.1 | PE | ID | | | 6 | NC |
| 16 | VIJAYAKUMAR | 60 | M | AP+VO | 6 | YES | NO | NO | NO | NO | NO | NO | 2098 | 2287 | FF | E | 8.9 | PE+FF+PN | AL | PN | ARDS,SEPTICEMIA | 16 | NFU |
| 17 | VADIVEL | 45 | M | AP | 5 | YES | NO | NO | NO | NO | YES | NO | 76 | 644 | PE | N | 7.8 | PE | AL | | HC | 7 | NC |
| 18 | ANANDHA | 35 | M | AP+VO | 4 | NO | NO | NO | NO | NO | NO | NO | 789 | 908 | N STUDY | N | 8.7 | PE | ID | | | 8 | NFU |
| 19 | ELIZABETH | 43 | F | AP+VO | 5 | NO | YES | NO | NO | NO | NO | NO | 978 | 1009 | BC | N | 9 | BC+PE | BI | | ARDS,CARDIO RESP ARREST | 2 | NC |
| 20 | VENKATESH | 38 | M | AP+VO | 2 | YES | NO | NO | NO | NO | NO | NO | 609 | 876 | N STUDY | N | 9.9 | PE | AL | | | 5 | NC |
| 21 | KARTHICK | 29 | M | AP+VO+AD | 5 | YES | NO | NO | NO | NO | YES | NO | 1807 | 2234 | PE+FF | N | 9.6 | PE+FF+PN | AL | PN+FF | | 10 | RAB |

| | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 22 | VEERABABU | 68 | M | AP+V0 | 5 | NO | YES | NO | NO | ERCP | NO | NO | 103 | 345 | N STUDY | E | 8.5 | PE | POST ERCP | | | 7 | NFU |
| 23 | KANNAN | 49 | M | AP+VO | 4 | YES | NO | NO | NO | NO | NO | NO | 76 | 509 | PE | E | 8.8 | PE | AL | | | 6 | RAB |
| 24 | KUMAR | 40 | M | AP+VO+AD | 6 | YES | NO | NO | NO | NO | NO | NO | 789 | 1265 | PS | N | 9 | PE+PS | AL | PN | SEPTICEMIA+ARF | 12 | NC |
| 25 | SARAVANAN | 40 | M | AP+VO | 3 | YES | NO | NO | NO | NO | NO | NO | 897 | 765 | PE | E | 9.7 | PE | AL | | | 12 | RAB |
| 26 | PANDHYALAKSHMI | 26 | F | AP+VO | 2 | NO | YES | NO | NO | NO | YES | NO | 765 | 986 | BC | N | 9.5 | BC+PE | BI | | | 7 | NFU |
| 27 | JEYARAMAMN | 48 | M | AP+VO+AD | 1 | YES | NO | NO | NO | NO | NO | NO | 95 | 756 | FF | E | 9.3 | PE+FF | AL | | | 5 | NC |
| 28 | DEVARAJ | 51 | M | AP+VO | 2 | YES | NO | NO | NO | NO | NO | NO | 98 | 806 | N STUDY | E | 8.9 | PE+PS | AL | PS | | 11 | NC |
| 29 | MALARVANNAN | 47 | M | AP+AD | 4 | YES | NO | NO | NO | NO | NO | NO | 2105 | 1256 | PE | E | 8.6 | PE+PN | AL | PN | SEPTICEMIA+ARF | 18 | NC |
| 30 | KUMAR | 38 | M | AP+VO | 2 | YES | NO | NO | NO | NO | NO | NO | 1098 | 1546 | PE | N | 9 | PE | AL | | | 7 | NC |
| 31 | SELVAM | 27 | M | AP+VO+AD | 3 | YES | NO | NO | NO | NO | NO | NO | 657 | 987 | FF | N | 9.3 | PE+FF | AL | | | 6 | RAB |
| 32 | SATHYADEV | 51 | M | AP+VO+AD | 2 | YES | NO | NO | NO | NO | NO | NO | 1123 | 1032 | PE+PS | E | 8.9 | PE+PS | AL | PS | SEPTICEMIA | 8 | NFU |
| 33 | ELUMALI | 39 | M | AP+VO | 4 | NO | NO | NO | NO | NO | YES | NO | 75 | 698 | BC | E | 8.1 | PE+BC | BI | | HC | 6 | RAB |
| 34 | MUNUSAMY | 48 | M | AP+VO+AD | 3 | YES | NO | NO | NO | NO | NO | NO | 108 | 907 | FF | E | 9 | PE+FF | AL | | | 5 | NC |
| 35 | DAS | 25 | M | AP | 3 | YES | NO | NO | NO | NO | NO | NO | 97 | 768 | N STUDY | N | 9.4 | PE | AL | | | 6 | NC |
| 36 | RANJITH | 25 | M | AP+VO | 4 | NO | NO | NO | NO | NO | YES | NO | 109 | 897 | N STUDY | N | 9.3 | PE | ID | | | 7 | NFU |
| 37 | JAYARAMAN | 45 | M | AP+VO | 2 | YES | NO | NO | NO | NO | NO | NO | 897 | 1098 | FF | E | 9.7 | PE+FF | AL | | | 6 | NC |
| 38 | MOORTHY | 55 | M | AP+VO+AD | 1 | YES | NO | NO | NO | NO | YES | NO | 1567 | 1897 | PE+PN | E | 7.9 | PE+PN | AL | PN | HC | 12 | RAB |
| 39 | KUMARAS | 50 | M | AP+VO | 1 | YES | NO | NO | NO | NO | NO | NO | 1366 | 1678 | PE+PS | E | 9.8 | PE+PS | AL | PS | PSEUDOCYST | 11 | RAB |
| 40 | RAJESH | 26 | M | AP | 2 | NO | NO | NO | NO | NO | NO | NO | 96 | 454 | BC | N | 8.9 | PE+BC | BI | | | 4 | NC |
| 41 | PANNOR | 45 | M | AP | 1 | YES | NO | NO | NO | NO | NO | NO | 109 | 768 | N STUDY | N | 8.8 | PE | AL | | | 7 | NC |
| 42 | SURESH | 23 | M | AP+VO | 2 | NO | NO | NO | NO | NO | NO | NO | 89 | 453 | N STUDY | N | 8.6 | PE | ID | | | 8 | NC |
| 43 | RAJA | 31 | M | AP+VO+AD | 3 | YES | NO | NO | NO | NO | NO | NO | 1985 | 2987 | PE+FF+PN | N | 8.9 | PE+FF+PN | AL | PN | ARDS | 15 | RAB |
| 44 | KUMAR | 50 | M | AP | 1 | YES | NO | NO | NO | NO | NO | NO | 110 | 567 | PE | E | 9.8 | PE | AL | | | 5 | NC |
| 45 | CHELLAMUTHU | 45 | M | AP+VO+AD | 4 | YES | NO | NO | NO | NO | NO | NO | 1009 | 1456 | PS | E | 9.6 | PE+PS | AL | PS | | 9 | RAB |

| | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 46 | CHEVAPATHY | 46 | M | AP+VO+AD | 2 | YES | NO | NO | NO | NO | YES | NO | 1987 | 2390 | PE+PN | N | 9 | PE+PN | AL | PN | SEPTICIMIA+ARF | 19 | NC |
| 47 | GIRI | 35 | M | AP+VO | 3 | NO | NO | NO | NO | NO | NO | NO | 83 | 322 | N STUDY | N | 8.8 | PE | ID | | | 7 | RAB |
| 48 | BHARATHI | 57 | M | AP | 5 | NO | NO | NO | NO | ERCP | NO | NO | 876 | 654 | N STUDY | E | 8.8 | PE | POST ERCP | | | 8 | NC |
| 49 | KUPPAN | 37 | M | AP+VO+AD | 4 | YES | NO | NO | NO | NO | NO | NO | 1876 | 2256 | FF+PN | N | 7.8 | PE+FF+PN | AL | PN+FF | HC | 17 | RAB |
| 50 | PAROSING | 54 | M | AP+VO | 3 | YES | NO | NO | NO | NO | NO | NO | 981 | 1009 | PE | E | 8.6 | PE | AL | | | 7 | NC |
| 51 | DHINAGARAN | 35 | M | AP+VO+AD | 5 | YES | NO | NO | NO | NO | NO | NO | 1123 | 1567 | PE+PN | N | 9 | PE+PN | AL | PN | | 11 | RAB |
| 52 | RAJA | 50 | M | AP+VO | 4 | YES | NO | NO | NO | NO | NO | NO | 679 | 905 | N STUDY | E | 9.7 | PE | AL | | | 5 | NC |
| 53 | ABDHUL | 26 | M | AP | 2 | NO | NO | NO | NO | NO | NO | NO | 112 | 541 | N STUDY | N | 9.4 | PE | ID | | | 7 | NC |
| 54 | KASIF | 20 | F | AP+VO+AD | 1 | NO | YES | NO | NO | NO | NO | NO | 907 | 905 | BC | N | 8.7 | BC+PE | BI | | | 7 | RAB |
| 55 | DEVA | 43 | M | AP+VO+AD | 1 | YES | NO | NO | NO | NO | NO | NO | 2108 | 2221 | PS | E | 8.9 | PE+PS | AL | PS | | 11 | NC |
| 56 | SENTHIL | 32 | M | AP | 1 | NO | NO | NO | NO | NO | NO | NO | 92 | 457 | PS | N | 9 | PE+PS | ID | PS | | 13 | RAB |
| 57 | MURUGAN | 47 | M | AP+VO | 2 | YES | NO | NO | NO | NO | NO | NO | 98 | 908 | PE | N | 9.9 | PE+BC | BI | | | 8 | NC |
| 58 | MOORTHY | 40 | M | AP+VO | 1 | NO | NO | NO | NO | NO | YES | NO | 112 | 896 | BC | E | 10 | BC+PE | AL | | | 5 | NFU |
| 59 | ARUL | 33 | M | AP+VO+AD | 3 | YES | NO | NO | NO | NO | NO | NO | 1908 | 2190 | FF+PN | N | 10 | PE+FF+PN | AL | PN | | 13 | RAB |
| 60 | JEYARAMAMN | 40 | M | AP+VO | 2 | NO | NO | NO | NO | NO | NO | NO | 98 | 650 | BC | N | 9.4 | BC+PE | BI | | | 9 | NC |
| 61 | MAHENDRAN | 34 | M | AP | 4 | NO | NO | NO | NO | NO | NO | NO | 567 | 1665 | BC | N | 9.9 | BC+PE | BI | | | 7 | NC |
| 62 | SUDHAKAR | 34 | M | AP+VO+AD | 2 | YES | NO | NO | NO | NO | YES | NO | 1256 | 1009 | PS | N | 8.9 | PE+PS | AL | PS | | 9 | RAB |
| 63 | BOOPATHY | 39 | M | AP+VO+AD | 3 | NO | NO | NO | NO | NO | NO | NO | 1123 | 1098 | BC+PS | E | 9 | PE+PS | BI | PS | | 9 | RAB |
| 64 | KANNIAMMAL | 36 | F | AP+VO+AD | 2 | YES | NO | NO | NO | NO | NO | NO | 875 | 1006 | PE | E | 8.9 | PE | AL | | | 7 | NC |
| 65 | SELVARAJ | 53 | M | AP+VO | 1 | YES | NO | NO | NO | NO | NO | NO | 458 | 1769 | PE | E | 9.6 | PE | AL | | | 7 | RAB |
| 66 | ANBAHAGAN | 51 | M | AP+VO | 3 | YES | NO | NO | NO | NO | NO | NO | 976 | 1096 | PE | E | 8.6 | PE | AL | | | 6 | RAB |
| 67 | MANOHARAN | 42 | M | AP | 2 | YES | NO | NO | NO | NO | NO | NO | 798 | 960 | N STUDY | E | 9 | PE | AL | | | 7 | NC |
| 68 | MANIKANNDAN | 58 | M | AP+VO | 4 | YES | NO | NO | NO | NO | NO | NO | 678 | 677 | PE | E | 9.8 | PE | AL | | | 16 | NC |
| 69 | LUKKA | 30 | M | AP+VO+AD | 2 | NO | YES | NO | NO | ERCP | YES | NO | 1123 | 1034 | FF+PN | N | 10 | PE+FF+PN | POST ERCP | PN+FF | | 15 | NFU |
| 70 | RAMESH | 41 | M | AP+VO | 1 | NO | NO | NO | NO | NO | NO | NO | 807 | 1098 | BC | E | 10 | PE+BC | BI | | | 7 | NC |
| 71 | VELU | 35 | M | AP+VO+AD | 1 | YES | NO | NO | NO | NO | NO | NO | 1765 | 1658 | PS | N | 9.8 | PE+PS | AL | PS | ARDS | 18 | RAB |
| 72 | VELAYDHAM | 50 | M | AP+VO+AD | 3 | YES | NO | NO | NO | NO | YES | NO | 764 | 1457 | N STUDY | E | 9.9 | PE | AL | | | 6 | NC |

| | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 73 | KANNAN | 49 | M | AP | 1 | NO | NO | NO | NO | NO | NO | NO | 98 | 698 | N STUDY | N | 9.4 | PE | ID | | | 7 | NC |
| 74 | PRIYA | 31 | F | AP | 2 | YES | NO | NO | NO | NO | NO | NO | 897 | 908 | PE | E | 9.4 | PE | AL | | | 8 | RAB |
| 75 | RAJA | 45 | M | AP+VO+AD | 3 | YES | NO | NO | NO | NO | NO | NO | 1087 | 1145 | FF+PN | E | 9.2 | PE+FF+PN | AL | PN+FF | | 14 | NC |
| 76 | ANTHONY | 40 | M | AP+VO+AD | 4 | YES | NO | NO | NO | NO | NO | NO | 1894 | 2098 | PE+PN | E | 9 | PE+PN | AL | PN | | 14 | NFU |
| 77 | KANNAN | 40 | M | AP+VO | 5 | NO | NO | NO | NO | NO | NO | NO | 58 | 346 | BC | N | 8.9 | BC+PE | BI | | | 9 | RAB |
| 78 | FRANCIS | 56 | M | AP+VO | 3 | YES | NO | NO | NO | NO | NO | NO | 1276 | 1156 | PE | E | 8.3 | PE | AL | | HC | 6 | NC |
| 79 | ANBANZHAGAN | 51 | M | AP | 2 | YES | NO | NO | NO | NO | NO | NO | 678 | 1786 | PE | E | 10 | PE | AL | | | 7 | NC |
| 80 | GOVINDASAMY | 48 | M | AP+VO | 3 | YES | NO | NO | NO | NO | NO | NO | 87 | 675 | N STUDY | E | 10 | PE | AL | | | 8 | NC |
| 81 | RAJA | 42 | M | AP+VO+AD | 1 | YES | NO | NO | NO | NO | YES | NO | 695 | 890 | PE | E | 8.9 | PE | AL | | | 6 | RAB |
| 82 | MANI | 57 | M | AP+VO | 3 | YES | NO | NO | NO | NO | NO | NO | 543 | 676 | PE | E | 8 | PE | AL | | HC | 8 | NC |
| 83 | MOHID | 45 | M | AP | 2 | YES | NO | NO | NO | NO | NO | NO | 54 | 543 | PE | E | 11 | PE | AL | | | 7 | NC |
| 84 | SAMBASIVAM | 32 | M | AP+VO+AD | 4 | YES | NO | NO | NO | NO | NO | NO | 457 | 1678 | PS | N | 11 | PE+PS | AL | PS | ARDS | 18 | RAB |
| 85 | ABDUL | 30 | M | AP+VO | 1 | YES | NO | NO | NO | NO | NO | NO | 564 | 698 | N STUDY | N | 9.5 | PE | AL | | | 7 | NC |
| 86 | RAJAN | 25 | M | AP+VO+AD | 3 | YES | NO | NO | NO | NO | NO | NO | 2654 | 2679 | PS | N | 9.3 | PE+PS | AL | PS | | 8 | RAB |
| 87 | MANOHARAN | 50 | M | AP | 2 | YES | NO | NO | NO | NO | NO | NO | 578 | 904 | PE | E | 9.1 | PE | AL | | | 9 | NC |
| 88 | UMA | 40 | F | AP+VO | 4 | NO | NO | NO | NO | NO | YES | NO | 456 | 1896 | BC | E | 9.7 | BC+PE | BI | | | 6 | NC |
| 89 | SUBRAMANI | 28 | M | AP+VO+AD | 2 | YES | NO | NO | NO | NO | YES | NO | 1789 | 1896 | PE+PN | N | 8.9 | PE+PN | AL | PN | | 11 | NFU |
| 90 | RAJAN | 25 | M | AP+VO+AD | 2 | YES | NO | NO | NO | NO | NO | NO | 64 | 890 | N STUDY | N | 8.4 | PE | AL | | HC | 9 | NC |
| 91 | KODHANDAN | 35 | M | AP+VO | 2 | NO | NO | NO | NO | NO | NO | NO | 110 | 586 | N STUDY | N | 8.6 | PE | ID | | | 7 | RAB |
| 92 | GUNASEKARAN | 53 | M | AP+VO | 1 | YES | NO | NO | NO | NO | NO | NO | 455 | 1765 | PE | E | 9.4 | PE | AL | | | 7 | NC |
| 93 | SURESH | 45 | M | AP | 4 | YES | NO | NO | NO | NO | NO | NO | 897 | 902 | PE | E | 9.4 | PE | AL | | | 8 | NC |
| 94 | RAMESH | 32 | M | AP+VO+AD | 3 | YES | NO | NO | NO | NO | NO | NO | 1765 | 1795 | FF+PN+PE | N | 9.3 | PE+PN+FF | AL | PN | | 13 | RAB |
| 95 | RAMAMMORTHY | 45 | M | AP+VO | 2 | YES | NO | NO | NO | NO | NO | NO | 890 | 674 | PE | E | 9.7 | PE | AL | | | 8 | NC |
| 96 | JEYARAMAN | 51 | M | AP+VO | 1 | YES | NO | NO | NO | NO | NO | NO | 48 | 765 | PE | E | 9.5 | PE | AL | | | 6 | NFU |
| 97 | ANTHONY | 40 | M | AP+VO+AD | 4 | YES | NO | NO | NO | NO | NO | NO | 46 | 896 | BC | E | 9.1 | BC+PE | BI | | | 5 | RAB |
| 98 | CHANDRAN | 20 | M | AP+VO | 1 | YES | NO | NO | NO | NO | NO | NO | 1960 | 2083 | N STUDY | N | 9.3 | PE | AL | | | 7 | NC |
| 99 | SILAMBARASAN | 27 | M | AP+VO | 2 | YES | NO | NO | NO | NO | NO | NO | 108 | 787 | PE | N | 8.4 | PE | AL | | HC | 8 | NFU |
| 100 | KRISHNAN | 46 | M | AP+VO+AD | 3 | YES | NO | NO | NO | NO | NO | NO | 1045 | 1437 | PS | E | 10 | PE+PS | AL | PS | | 7 | NC |

# INSTITUTIONAL ETHICS COMMITTEE
## MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax: 044 25363970

## CERTIFICATE OF APPROVAL

To

**Dr.K.J.VIGNESVARAN,**
Post Graduate,
Institute of General Surgery,
Madras Medical College,
Chennai - 600 003.

Dear **Dr.K.J.VIGNESVARAN,**

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled **"A Study of Etiological analysis of acute pancreatitis and their outcome in patients admitted at RGGGH"** No.490614.

The following members of Ethics Committee were present in the meeting held on 03.06.2014 conducted at Madras Medical College, Chennai-3.

| | | |
|---|---|---|
| 1. | Dr. C.Rajendran, M.D, | -- Chairperson |
| 2. | Prof. Kalaiselvi, M.D,<br>Vice Principal, MMC, Ch-3 | -- Member Secretary |
| 3. | Prof. Nandhini, M.D,<br>Inst. of Pharmacology, MMC, Ch-3 | -- Member |
| 4. | Prof.G.Muralidharan, M.S,<br>Prof & HOD General Surgery, MMC, Ch-3 | -- Member |
| 5. | Prof.V.Padmavathi, M.D,<br>I/c. Director of Pathology, MMC, Ch-3 | -- Member |
| 6. | Thiru. S. Govindasamy, BA., BL | -- Lawyer |
| 7. | Tmt.Arnold Saulina, MA MSW | -- Social Scientist |
| 8. | Thiru.S.Ramesh Kumar,<br>Administrative Officer, MMC, Ch-3. | -- Lay Person |

We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLE

A Dissertation on

# "A STUDY OF ETIOLOGICAL ANALYSIS OF ACUTE PANCREATITIS AND THEIR OUTCOME IN PATIENTS ADMITTED AT RGGGH"

Dissertation submitted to

**THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERISTY**

**CHENNAI**

with partial fulfilment of the regulations

for the Award of the degree

**M.S. (General Surgery)**

Branch – I

**MADRAS MEDICAL COLLEGE ,**